CLINICAL PROFILE OF ACUTE KIDNEY INJURY DUE TO MEDICAL DISORDERS IN CHENNAI

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CERTIFICATE

This is to certify that the dissertation titled **"CLINICAL PROFILE OF ACUTE KIDNEY INJURY DUE TO MEDICAL DISORDERS IN CHENNAI"** is the bonafide original work of **Dr. P. VIJAI ANANTH** in partial fulfillment of the requirements for M.D. Branch – I (General Medicine) Examination of the Tamilnadu DR. M.G.R Medical University to be held in MARCH 2009. The period of study was from September 2007 to September 2008

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DECLARATION

I, **Dr. P. VIJAI ANANTH** hereby solemnly declare that the dissertation titled **"CLINICAL PROFILE OF ACUTE KIDNEY INJURY DUE TO MEDICAL DISORDERS IN CHENNAI"** was done by me at Government Stanley Medical College and hospital during September 2007 to September 2008 under the guidance and supervision of my unit chief Prof. S. SUNDAR.

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CONTENTS

INTRODUCTION

Acute kidney injury (AKI) is a protean syndrome of varied severity. It is characterized by a rapid (hours to weeks) decline in the glomerular filtration rate (GFR) and retention of nitrogenous waste products such as blood urea nitrogen (BUN) and creatinine[1,2]. In recent years, it has been recognized that the time – honored term Acute Renal Failure fails to adequately describe what is a dynamic process extending across initiation, maintenance and recovery phases, each of which may be of variable duration and severity. The term Acute Renal Failure suggests that the syndrome is dichotomous and places an undue emphasis on whether or not renal function has overtly failed. This believes the now well-established fact that even mild decrements in glomerular filtration may be associated with adverse clinical outcomes[3,4]. The alternative proposed term acute kidney injury has much to recommend it, perhaps better captures the diverse nature of this syndrome and has entered into widespread clinical use.

AKI may occur in someone either with previously normal renal function or as an acute and anticipated deterioration in function in the setting of previously established chronic kidney disease. The etiology and outcomes of AKI is heavily influenced by the circumstances in which it occurs, such as whether it develops in the community or in the hospital. It is similarly important to distinguish whether the

kidney injures occurs as an isolated process, which is more common in community acquired AKI, or if it occurs as part as a more extensive multiorgan syndrome[5].

In 2004, Acute Dialysis Quality Initiative (ADQI) group, International Society of Nephrology (ISN), National Kidney Foundation (NKF) and American Society of Nephrology (ASN) met and proposed the term ‘Acute Kidney Injury. AKI generally defined as:

‘*an abrupt and sustained decrease in kidney function*’

Acute Dialysis Quality Improvement Initiative (ADQI) has proposed a new definition of AKI, that has been widely endorsed and is increasingly being used in keeping with the spectrum of changes seen in AKI, the diagnostic classification scheme was developed, this scheme is referred to by the acronym RIFLE, and includes three levels of renal dysfunction of increasing severity, namely. Risk of renal dysfunction, injury to the kidney and failure of kidney functions, and two outcome categories: Loss of function. And End stage kidney disease. Renal dysfunction is defined in terms of a rise in creatinine or a reduction in urine output, the more severe of the two criteria being selected. When achieved designation results from urine output criteria a subscript "o" is added e.g. RIFLE-Fo. Similarly, a subscript of "c" is used to denote the presence of preexisting renal disease[6].

RIFLE classification scheme for Acute Kidney Injury[6]

	GFR criteria	Urine output criteria	
risk	Increased Serum Creatinine x 1.5 GFR decrease >25%	UO<.5 ml/ kg /h X 6hr	High Sensitivity
injury	Increased Serum Creatinine x 2 or GFR decrease > 50%	UO< .5 ml/ kg / h X 12 hr	
Failure	Increased Serum Creatinine x 3 GFR decrease > 75% Or Serum Creatinine $\leq$ 4 mg / dl Acute rise >.5 mg/dl	UO < .3 ml/ kg / h X 24 hr or Anuria x 12 hrs	

Loss	Persistent AKI = complete loss of Kidney function > 4 weeks	High specificity
ESKD	End Stage Kidney Disease (> 3months)	

RIFLE is also useful at predicting outcome data including recovery of renal function, length of hospital stay, renal replacement requirement and in-hospital mortality.

In 2004, Acute Kidney Injury Network (AKIN) formed .AKIN proposed a diagnostic criteria for the definition AKI:

"An abrupt (within 48 hours) reduction in kidney function currently defined as an absolute increase in serum creatinine of more than or equal to 0.3 mg/dl (≥ 26.4 μmol/l), a percentage increase in serum creatinine of more than or equal to 50% (1.5-fold from baseline), or a reduction in urine output (documented oliguria of less than 0.5 ml/kg per hour for more than six hours)."

Data has emerged recently that suggests smaller increases in Serum Creatinine than those considered in RIFLE criteria may be associated with adverse outcomes. So **AKIN proposed a new classification/staging system.**

Classification/staging system for acute kidney injury

Stage	Serum creatinine criteria	Urine output criteria
1	Increase in Serum Creatinine of ≥ 0.3 mg/dl (≥26.4 μmol/l) or to ≥ 150% to 200% (1.5 to 2 fold) from baseline	Less than 0.5 ml/kg per hr for more than 6 hrs
2	Increase in Serum Creatinine to more than 200% to 300% (> 2 – 3 fold) from baseline	Less than 0.5 ml/kg per hr for more than 12 hrs
3	Increase in Serum Creatinine to more than 300% (>3 fold) from baseline or Serum Creatinine ≥4.0 mg/dl [≥354 μmol/l] with an acute increase of at least 0.5 mg/dl [44 μ/l]	Less than 0.3 ml/kg per hr or anuria 12 hrs

A major challenge in the investigation and management of AKI is timely recognition of the syndrome. It remains difficult to easily and reliably measure rapid changes in the GFR. Although the severity in decline in GFR correlates with the onset of oliguria, the latter is insensitive marker of the syndrome because many subjects with severe renal failure remain non oliguric. In AKI, there is a poor agreement between serum creatinine and GFR, at least until a serum creatinine steady state is reached and even then , the absolute rise in serum creatinine must take into account differences in creatinine generation rates[7].

AIMS AND OBJECTIVES

1) To study about the etiological profile of acute kidney injury
2) To apply the RIFLE criteria in acute kidney injury patients admitted to the medical wards and to confirm the significance
3) To study about the prognosis and outcomes of acute kidney injury

REVIEW OF LITERATURE

Acute Kidney Injury of course must have followed on many major traumas and tragedies throughout the history, but had not been noted until the 20th century. The first report of fatal Acute Kidney Injury is accreditated to Hackradt, a German pathologist in 1917 and was based on soldiers who sustained severe traumatic injuries. The concept of Acute Kidney Injury on a previously normal kidney was better understood during and after second worldwar. In 1941 Bywaters and Beall described 'crush kidney' syndrome in victims of London Blitz[9]. Subsequent studies showed acute, potentially reversible failure of renal function, associated with histological findings of acute tubular necrosis, could also due to other causes such as mismatched blood transfusion abortion, cardiovascular collapse, sepsis and a variety of nephrotoxic substances.

In recent years, new causes which have been reported include Hantaan virus, ingestion of raw fish gallbladder, quinine hypersensitivity, chewing match heads, Ectasy, HIV infection, physical torture, inhalation of mycotoxins, gelatin infusion, and herbal medicine[8].

Nash *et al,* 2002[61] using same AKI defining criteria at a different institution, reported frequency of AKI in hospitalised pts had increased to 7.2%.In-hospital mortality rate was 19.4%. Major causes were decreased renal perfusion (39%),

nephrotoxin administration (16%), contrast administration (11%), major surgery (9%)

Liano *et al*, 1996 Madrid[62] did study in 13 centres covering 4.2 million people 748 episodes of AKI during 200,464 admissions (0.37%). Overall incidence of AKI was 209 cases per million population. Frequent causes: ATN (45%), prerenal (21%), acute-on-chronic kidney disease (12.7%), obstructive (10%). Mortality was much higher in AKI pts (45%) to other pts admitted (5.4%)

Ostermann M, Chang RW: CCM 2007[63] showed AKI is an independent risk factor for death**.** Patients don't just die with AKI they die because of AKI.

They detected the following:

- RIFLE class F have a mortality of 57%
- RIFLE class I 45%
- RIFLE class R 21 %

Compared to 8.4% of patients without AKI

The aetiology of AKI varies from place to place within the country. Acute diarrhoeal disease is the commonest cause of AKI. The incidence of snake bite induced AKI is higher in North India, while leptospirosis AKI commonly encountered in Kerala and Chennai. Malarial AKI is common in Eastern India.

INCIDENCE

AKI complicates approximately 5-7% of hospital admissions[10] and up to 30% of admissions to intensive care units[11]. Retention of nitrogenous waste products, oliguria (urine output <400 mL/d contributing to extracellular fluid overload), and electrolyte and acid base abnormalities are frequent clinical features. AKI is usually asymptomatic and diagnosed when biochemical monitoring of hospitalized patients reveals a new increase in blood urea and serum creatinine concentrations.

For purposes of diagnosis and management, AKI has been divided into three categories.

1. Disease characterized by renal hypoperfusion in which the integrity of renal parenchymal tissue is preserved (prerenal states) (~55%);

2. Diseases involving renal parenchymal tissue (intrarenal AKI or intrinsic AKI(~40%); and

3. Diseases associated with acute obstruction of urinary tract (post renal or obstructive AKI) (~5%).

AKI is often considered to be reversible, although a return to baseline serum creatinine concentrations postinjury might not be sufficiently sensitive to detect clinically significant irreversible damage that may ultimately contribute to chronic kidney disease. AKI is associated and significant in-hospital morbidity and

mortality, the latter in the range of 30-60%, depending on the clinical setting and presence or absence of nonrenal organ system failure.

CLASSIFICATION AND MAJOR CAUSES OF ACUTE KIDNEY INJURY[64]:

PRERENAL AKI

1) Hypovolemia

a) Increased extracellular fluid losses: hemorrhage

b) Gastrointestinal fluid loss: vomiting, diarrhea, enterocutaneous fistula

c) Renal fluid loss: diuretics, osmotic dieresis, hypoadrenalism, nephrogenic diabetes insipidus

d) Extravascular sequestration: burns, pancreatitis, severe hypoalbuminemia (hypoproteinemia)

e) Decreased intake: dehydration, altered mental status

2) Altered renal hemodynamics resulting in hypoperfusion

a) Low cardiac output state: diseases of the myocardium, valves, and pericardium (including tamponade); pulmonary hypertension or massive pulmonary embolism leading to right and left heart failure; impaired venous return (eg. Abdominal compartment syndrome or positive pressure ventilation)

b) Systemic vasodilation: sepsis, anti-hypertensives, afterload reducers, anaphylaxis

c) Renal vasoconstriction: hypercalcemia, catecholamines, calcineurin inhibitors, amphotericin B

d) Impairment of renal autoregulatory responses: cyclooxygenase inhibitors (NSAIDs), ACE Inhibitors[15], or AT II receptor blockers

e) Hepatorenal syndrome

INTRINSIC AKI

1) Renovascular obstruction (bilateral or unilateral in the setting of one kidney)

a) Renal artery obstruction: atherosclerotic plaque, thrombosis, embolism, dissecting aneurysm, large vessel vasculitis

b) Renal vein obstruction: thrombosis or compression

2) Diseases of the glomeruli or vasculature

a) Glomerulonephritis or vasculitis

b) Other: thrombotic microangiopathy, malignant hypertension, collagen vascular disease (systemic lupus erythematosus, scleroderma), DIC, preeclampsia

3) Acute tubular necrosis

a) Ischemia: causes are the same as for prerenal AKI, but generally the insult is more severe and/or more prolonged

b) Infection, with or without sepsis syndrome

c) Toxins: 1. Exogenous**:** radiocontrast, calcineurin inhibitors, antibiotics (eg. Amphotericin B), ethylene glycol,

2. Endogenous: rhabdomyolysis, hemolysis

4) Interstitial nephritis

a) Allergic: antibiotics (betalactams, sulfonamides, quinolones, rifampin), NSAIDs, diuretics, other drugs

b) Infection: pyelonephritis (if bilateral)

c) Infiltration: lymphoma, leukemia, sarcoidosis

d) Inflammatory, non-vascular: Sjogren's syndrome, tubulointerstitial nephritis with uveitis

5) Intratubular obstruction

a) Endogenous: myeloma proteins, uric acid (tumor lysis syndrome), systemic oxalalosis

b) Exogenous: acyclovir, gancyclovir, methotrexate, indinavir

POSTRENAL AKI (obstruction)

1) ***Ureteric (bilateral or unilateral in the case of one kidney):*** calculi, blood clots, sloughed papillae, cancer, external compression (eg. Retroperitoneal fibrosis)

2) ***Bladder neck:*** neurogenic bladder, prostatic hypertrophy, calculi, blood clots, cancer.

ETIOLOGY AND PATHOPHYSIOLOGY

PRERENAL AKI

The most common form of AKI is prerenal AKI, which occurs in the setting of renal hypoperfusion. Prerenal AKI is generally reversible when perfusion pressure is restored. By definition, renal parenchymal tissue is not damaged. More severe or prolonged hypoperfusion may lead to ischemic injury, often termed acute tubular necrosis, or ATN. Thus prerenal AKI and ischemic ATN fall along a spectrum of manifestations of renal hypoperfusion. Prerenal AKI complicate any disease that induces hypovolemia, low cardiac output, systemic vasodilatation, or selective intrarenal vasoconstriction.

Hypovolemia leads to a fall in mean systemic arterial pressure, which is detected as reduced stretch by arterial (e.g., carotid sinus) and cardiac baroreceptors. In turn, this triggers a coordinated series of neurohormonal

responses that aim to restore blood volume and arterial pressure. These include activation of the sympathetic nervous system and renin-angiotensin-aldosterone system, as well as release of arginine vasopressin[12]. Relatively "nonessential" vascular beds (such as the musculocutaneous and splanchnic circulations) undergo vasoconstriction in an attempt to preserve cardiac and cerebal perfusion pressure. In additional salt loss through sweat glands is inhibited, and thirst and salt appetite are stimulated. Renal salt and water retention also occur.

In states of mild hypoperfusion, glomerular perfusion and the filtration fraction are preserved through several compensatory mechanisms. In response to the reduction in perfusion pressure, stretch receptors in afferent arterioles trigger afferent arteriolar vasodilatation through a local myogenic reflex (autoregulation). Angiotensin II increases biosynthesis of vasodilator prostaglandins (e.g., prostaglandin E2 and prostacyclin), also resulting in afferent arteriolar vasodilation. In addition, angiotensin II induces preferential constriction of efferent arterioles. As a result, the fraction of plasma flowing through glomerular capillaries that is filtered is increased (filtration fraction), intra glamerular pressure is maintained, and GFR is preserved. With more severe hypoperfusion, these compensatory responses are overwhelmed and GFR falls, leading to prerenal AKI[13,14].

Autoregulatory dilatation of afferent arterioles allows for maintenance of GFR despite systemic hypotension; however, when hypotension is severe or prolonged, these autoregulatory mechanisms fail, resulting in a precipitous decline

in GFR. Lesser degrees of hypotension may provoke prerenal AKI in those at risk: the elderly and patients with diseases that affect the integrity of afferent arterioles (e.g., hypertensive nephrosclerosis, diabetic vasculopathy and other forms of occlusive including atherosclerotic renovascular disease). In addition, drugs that interfere with adaptive responses to hypoperfusion may convert compensated renal hypoperfusion into overt prerenal AKI or ATN. Pharmacologic inhibitors of renal prostaglandin biosynthesis (nonsteridal anti-inflammatory drugs[16] (NSAIDs) or angiotensin-converting enzyme inhibitors[15] (ACEI) and angiotensin II receptor blockers (ARBs) are major culprits. While NSAIDs do not compromise GFR in healthy individuals, these medications may perecipitate prerenal AKI in patients with volume depletion or in those with chronic kidney disease (in whom GFR is maintained, in part, through prostaglandin mediated hyperfiltration by the remaining fuctional nephrons). ACE inhibitors should be used with special care in patients with bilateral renal anery stenosis or unilateral stenosis in a solitary functioning kidney. In these settings, glomerular perfusion and filtration may be exquisitely dependent on the actions of angiotensin II. Angiotensin II preserves GFR in these circumstances by raising systemic arterial pressure and by triggering selective constriction of efferent arterioles. ACE inhibitors and ARBs blunt these responses and can precipitate AKI.

INTRINSIC AKI

Intrinsic cause of AKI can be conceptually divided based on the predominant compartment of the kidney that is affected:

(1) Ischemic or nephrotoxic tubular injury

(2) Tubulointerstitial diseases

(3) Diseases of the renal microcirculation and glomeruli, and

(4) Diseases of larger renal vessels.

Ischemia and nephrotoxins classically induce acute tubular injury. Although many patients with ischemic or nephrotoxic AKI do not have morphologic evidence of cellular necrosis, this disease is often referred to as acute tubular necrosis, or ATN. More recently, because of the important role of sublethal injury to tubular epithelial and other renal cells (e.g., endothelial cells) in the pathogenesis of this syndrome, the term acute kidney injury (AKI)has been proposed.

ETILOLOGY AND PATHOPHYSIOLOGY OF ISCHEMIC ATN

Prerenal AKI and ischemic ATN are part of a spectrum of manifestations of renal hypoperfusion[17], In its most extreme form, ischemia leads to bilateral renal cortical necrosis and irreversible renal failure. ATN differs from prerenal AKI in that the renal tubular epithelial cells are injured in the latter. ATN occurs most frequently in patients undergoing major cardiovascular surgery of suffering severe

trauma, hemorrhage, sepsis, and/or volume depletion[18,19,20]. Patients with other risk factors for AKI (e.g., exposure to nephrotoxins or preexisting chronic kidney disease) are at increased risk for ATN. Recovery typically takes 1-2 weeks after normalization of renal perfusion, as it requires repair and regeneration of renal cells.

The course of ischemic ATN is typically characterized by four phases: initiation, extension, maintenance, and recovery. These phases are often preceded by a period of prerenal azotemia.

During the inititation phase (lasting hours to days), GFR declines because (1) glomerular ultrafiltration pressure is reduced as renal blood flow falls, (2) the flow of filtrate within tubules is obstructed by casts comprised of shed epithelial cells and necrotic debris, and (3) there is backleak of glomerular filtrate through injured tubular epithelium. Ischemic injury is most prominent in the S3 segment of the proximal tubule and the medullary portion of the thick ascending limb of the loop of Henle. These segments of the tubule are particularly sensitive to ischemia because of high rates of active (ATP-dependent) solute transport and location in the outer medulla, where the partial pressure oxygen is low, even under basal conditions, Cellular ischemia results in ATP depletion, inhibition of active sodium transport, cytoskeletal disruption, loss of cell polarity, cell-cell and cell-matrix attachment, and Oxygen free- radical formation[26]. Renal injury may be limited by restoration of renal blood flow during this period. If severe, cell injury results in apoptosis or necrosis.

The extension phase follows the initiation phase and is characterized by continued ischemic injury and inflammation. It has been proposed that endothelial damage (resulting in vascular congestion) contributes to both of these processes. During the maintenance phase (typically 1-2 weeks), GFR stabilizes at its nadir (typically 5-10 mL/min), urine output is lowest, and uremic complications may arise. Its not clear why the GFR remains low during this phase, despite correction of systemic hemodynamics. Proposed mechanisms include persistent intrarenal vasoconstriction and medullary ischemia trigger dysregulated release of vasoactive mediators from injured endothelial cells, congestion of medullary blood vessels, and reperfusion injury induced by reactive oxygen species and inflammatory mediators released by leukocytes or renal parenchymal cells. In addition, epithelial injury may contribute to persistent intrarenal vasoconstriction through tubuloglomerular feedback. Specialized epithelial cells in the macula densa region of distal tubules detect increases in distal salt delivery that occur as a consequence of impaired reabsorption by more proximal nephron segments. Macula densa cells, in turn, stimulate constriction of adjacent afferent arterioles by poorly defined mechanism and further compromise glomerular perfusion and filtration, thereby contributing to vicious circle.

The recovery phase is characterized by tubular epithelial cell and regeneration as well as a gradual return of GFR toward premorbid levels. The recovery phase may be complicated by a marked diuretic phase due to delayed

recovery of epithelial cell function (solute water reabsorption) relative to glomerular filtration.

ETIOLOGY AND PATHOPHYSIOLOGY OF NEPHROTOXIC AKI

Nephrotoxic ATN may complicate exposure to many structurally diverse pharmacologic agents. With most nephrotoxins, the incidence of AKI is in the elderly and in patients with preexisting chronic kidney, true or "effective" hypovolemia, or concomitant exposure to toxins.

Radiocontrast agents[21], cyclosporine, and tacrolimus cause kidney injury through intrarenal vasoconstriction. Consequently, ATN in asssociation with these medications is characterized by an acute fall in renal blood flow and GFR, a relatively benign urine sediment, and a low fractional excretion of sodium. Severe cases may show clinical or pathologic evidence of tubular cell necrosis. Contrast nephropathy is also thought to result from the generation of reactive oxygen species that are directly toxic to renal tubular cells. Contrast nephropathy classically presents as an acute(onset within 24-48 hrs) but reversible (peak 3-5 days, resolution with in week) rise in blood urea nitrogen and serum creatinine. Contrast nephropathy is most common in individuals with preexisting chronic disease, diabetes mellitus, congestive heart failure, hypovolemia or multiple myeloma. The type (low vs. isoosmolar contrast) and dose of contrast also influence the likelihood of injury associated with administration.

Antibiotics and anticancer drugs typically cause ATN through direct toxicity to the tubular epithelial cells and/or intratubular obstruction. AKI Complicates 10-30% of courses of aminoglycoside antibiotics[23]. Aminoglycosides accumulate in renal tubular epithelial cells, where they cause oxidative stress and cell injury; thus, AKI usually occurs after several days of aminoglycoside therapy. Damage may occur in both the proximal and distal tubule; defects in the distal tubule may result in decreased concentrating ability. Amphotericin B causes dose-related AKI through intranenal vasoconstriction and direct toxicity to proximal tubule epithelium. Newer (liposomal) formulations of amphotericin B may be associated with less nephrotoxicity. Acyclovir may precipitate in renal tubules and cause Acute Kidney Injury. Foscarnet and pentamidine are less commonly prescribed antimicrobials also frequently associated with Acute Kidney Injury. Cisplatin[22] and carboplatin, like the aminoglycosides, are accumulated by proximal tubule cells and typically provoke AKI after 7-10 days of exposure, typically in association with potassium and magnesium wasting. Ifosphamide administration may lead to hemorrhagic cystitis, manifested by hematuria, as well as acute and chronic renal failure. Type II renal tubular acidosis (Fanconi syndrome) often accompanies ifosphamide-associated AKI.

Endogenous nephrotoxins include calcium, myoglobin, hemoglobin, urate, oxalate, and myeloma light chains[24]. Hypercalcemia can compromise GFR, predominantly by inducing intrarenal vasoconstriction as well as volume depletion

from obligate water loss. Both rhabdomyolysis and hemolysis can induce AKI. Common causes of rhabdomyolysis include traumatic crush injury, acute muscle ischemia, prolonged seizure activity, excessive exercise, heat stroke or malignant hyperthermia, and infectious or metabolic disorders (e.g., hypophosphatemia, severe hypothyrodisim). AKI due to hemolysis is relatively rare and is observed following blood transfusion reactions. It has been postulated that myoglobin and hemoglobin promote intrarenal oxidative stress, resulting in injury to tubular epithelial cells and inducing intratubular cast formation. In addition, cell-free hemoglobin and myoglobin are potent inhibitors of nitric oxide bioactivity and may trigger intrarenal vasoconstriction and ischemia. Hypovolemia or acidosis may further promote intratubular cast formation. Intratubular casts containing filtered immunoglobulin light chains and other proteins (including Tamm-Horsfall protein produced by thick ascending limb cells) cause AKI in patients with multiple myeloma (myeloma cast nephropathy)[25]. Light chains are also directly toxic to tubule epitherlial cells. Intratubular obstruction is an important cause of AKI in patients with severe hyperuricosuria or hyperoxaluria. Acute uric acid nephropathy can complicate the treatment of selected lymphoproliferative or myeloproliferative disorders (e.g., Burkitt's lymphoma, acute myelogenous leukemia), especially after the administration of chemotherapy, resulting in increased cell lysis ('tumor lysis syndrome').

OTHER CAUSES OF INTRINSIC AKI

Virtually any pharmacologic agent may trigger allergic interstitial nephritis, which is characterized by infiltration of the tubulointerstitum by granulocytes (typically but not invariably eosinophils), macrophages, and/or Iymphocytes and by interstitial edema. The most common offenders are antibiotics (e.g., penicillins, cephalosporins, quinolones, sulfonamides, rifampin) and NSAIDs.

Patients with advanced atherosclerosis can develop AKI after manipulation of the aorta or renal arteries during surgery or angiography, following trauma, or, rarely, spontaneously (atheroembolic AKI). Cholesterol crystals embolize to the renal vasculature, lodge in small- and medium-sized arteries, and incite a giant cell and fibrotic reaction in the vessel wall with narrowing or obstruction of the vessel lumen. Atheroembolic AKI is often associated with hypocomplementemia and eosinophiluria, and it is frequently irreversible. The acute glomerulonephritides and immune-mediated diseases characterized by proliferative or crescentic glomerular inflammation (glomerulonephritis).

POSTRENAL AKI

Urinary tract obstruction accounts for fewer than 5% of cases of hospital-acquired AKI. Because one kidney has sufficient reserve to handle generated nitrogenous waste products, AKI from obstruction requires obstruction to urine flow between the external urethral meatus and bladder neck, bilateral ureteric

obstruction, or unilateral ureteric obstruction in a patient with one functuioning kidney or with significant preexisting chronic kidney disease. Bladder neck obstruction is the most common cause of postrenal AKI and is usually due to prostatic disease (e.g., hypertrophy, neoplasia, or infection), neurogenic bladder, or therapy with anticholinergic drugs. Less common causes of acute lower urinary tract obstruction include blood clots, calculi, and urethritis with spasm. Ureteric obstruction may result from intraluminal obstruction (e.g., calculi, blood clots, sloughed renal papillae), infiltration of the ureteric wall (e.g., neoplasia), or external compression (e.g., retroperitoneal fibrosis, neoplasia or abscess, inadvertent surgical ligature). During the early stages of obstruction (hours to days), continued glomerular filtration leads to increased intraluminal pressure upstream to the site of obstruction. As a result, there is gradual distention of the proximal ureter, renal pelvis, and calyces and a fall in GFR.

AKI IN SPECIAL SETTINGS

Neonatal AKI is commonly due to *immature defence* system and dehydration. In older children AKI is due to acute diarrhoeal disease, post infective glomerulonephritis, hemolytic uremic syndrome, and nephritic syndrome.

In elderly people incidence of AKI is high. It may be due to 1) aging kidney is less able to cope up with rapid hemodynamic alterations and rapid changes in salt and water. 2) High incidence of comorbid illnesses. 3) The elderly people consume too many medications in comparing with other age groups.

Acute kidney injury in a patient with cancer

Most AKI in patiens with cancer is due to either prerenal AKI - often induced by vomiting and diarrhea in the presence of NSAIDS use -or hypercalcemia. Intrinsic AKI can be triggered by chemotherapeutic agents or by the products of tumor lysis[51]. Renal parenchymal invasion by the solid and hematological cancers occurs in 5% to 10% of autopsy studies but it s rarely of clinical significance. AKI consequent to leukemia with infiltration of the kidney parenchyma typically presents with heamturia, protienuria and enlarged kidneys on ultrasound imaging. The diagnosis is a important one because the AKI may respond to chemotherapeutic intervention.

The tumour lysis syndrome is characterized by AKI associated with hyperuricemia, hyperphosphatemia and hypocalcemia. It occurs in most often following initiation of chemotherapy in patients with poorly differentiated lymphoproliferative malignancies, particularly the acute leukemias. It occasionally occurs spontaneously or in patients with solid organ tumors. AKI is triggered by direct tubular injury/ obstruction by uric acid and calcium phosphate crystals. Less common cause of AKI include tumor associated glomerulonephritis or a thrombotic microangiopathy(TMA) induced by drugs or irradiation. In regard to the latter, chemotherapy-associatd TMA is a well-recognised complications of several chemotherapeutic agents of which mitomycin C and gemcytabine are pre-eminent[51].

AKI in association with multiple myeloma carries a wide differential diagnosis with includes in decreasing order of frequency hypovolemia, myloma cast nephropathy, sepsis, hypercalcemia,ATN induced by drugs or tumor lysis during therapy, light chain deposition disease, cryoglobulinemia, hyperviscosity syndrome, plama cell infiltration and vascular amyloidosis[25].

Acute kidney injury in pregnancy

In early pregnancy, ATN induced by nephrotoxic abortificients is still a relatively common cause of AKI in developing countries but it is rarely seen in developed countries. Ischemic ATN, severe toxemia of pregnancy and postpartum HUS and TTP are the most common causes later in pregnancy. Ischemic ATN is usually provoked by postpartum hemorrhage or abruptio placentae and less commonly by amniotic fluid embolism or sepsis[52]. Glomerular filtration is usually a normal, mild and moderate pre- eclampsia. However AKI may complicate severe disease. In this setting, AKI is typically transient and found in association with intrarenal vasospasam, marked hypertension and neurologic abnormalities. A variant of pre eclampsia, the HELLP syndrome(hemolysis, Elevated liver enzymes, Low platelets), is characterized by an initial benign course that can be rapidly deteriorate with the development of a thrombotic microangiopathy characterized by hemolysis and derangement of coagulation, and hepatic and renal function. Immediate delivery of fetus is indicated in such case .This presentation

contrast with that of postpartum thrombotic microangiopathy, which typically occurs against a background of normal pregnancy is characterized by postpartum thrombocytopenia,microangiopathic anemia, and normal prothromin and partial thromboplastin times and frequently causes long term- impairment of renal function.

Acute kidney injury in association with pulmonary diseae[53]

The co existence of AKI and pulmonary disease (pulmonary renal syndrome) classically suggests a diagnosis of good pasture syndrome, Wegener granulomatosis, or other vasculitides. The detection of circulating antineutrophil cytoplasmic antibodies, anti glomerular basement membrane antibodies or hypocomplementinemia can be useful in differentiation of these diseases. Although the urgent need for definitive diagnosis and treatment may mandate a lung or renal biopsy. Several toxic ingestions And infections may also cause simultaneous pulmonary and renal injury that mimics a vasculitis process. Furthermore intrinsic renal and postrenal AKI of any cause may be complicated by secondary hypervolemia and pulmonary edema and severe lung disease may compromise cardiac output and induce prerenal AKI.

Acute kidney injury in association with chronic liver disease[54]

The differential diagnosis for AKI in association with liver disease is similarly large. In chronic liver disease, causes of AKI include volume depletion, gastrointestinal hemorrhage, sepsis, nephrotoxins (antibiotics, radiocontrast)and

the HRS. The term HRS is usually reserved for a syndrome of irreversible AKI that usually complicates advanced cirrhosis; however this syndrome has been described in association with fulminant viral and alcoholic hepatitis[54]. The syndrome is characterized by renal failure and disturbed regulation of circulatory function. The latter is characterized by intense intrarenal vasoconstriction, whereas in the extrarenal circulation, arterioral vasodilatation triggers a reduction in total peripheral vascular resistance and a decrease in effective systemic circulatory volume despite an expanded total extra cellular fluid. Most patients have clinical evidence of advanced cirrhosis. HRS almost certainly represents the terminal stage of a hypoperfusion state that begins early in the course of chronic liver disease. The pathogenic mechanisms for the dramatic hemodynamic alternations are incompletely understood. In the early stages of HRS. Arterial under filling is thought to trigger activation of the rennin angiotensin and sympathetic nervous systems.

Two subtypes of HRS have been described**; type1** is characterized by a rapid onset of renal failure with a doubling of serum creatinine to greater than 2.5mg/dl or a 50% reduction in GFR to less than 20 mL/min over a 2-week period. This subtype is characterized by a fulminant course with oliguria, encephalopathy,marked hyperbilirubinemia and death usually within 1 month of presentation. **Type II HRS** is typifies in a more indolent course with a stable reduction in GFR accompanying diuretic resistant ascites and avid sodium retention. The diagnosis of HRS is one of

exclusion. other diagnoses that must be entertained in the patient with AKI and liver disease include prerenal AKI due to gastrointestinal losses, drug toxicity, combined hepatitis and tubulointerstitial nephritis induced by drugs or infectious agents and multiorgan involvement in vasculitides(hepatitis C- induced cryoglobulinemia). The BUN and serum creatinine values are characterized deceptively low, despite marked impairment of GFR, because of impaired urea generation and co existing muscle wasting. The urinary findings include a benign sediment and a low FENa, the most common precipitant of the HRS in patients with compensatd cirrhosis is spontaneous bacterial peritonitis.

DIAGNOSTIC EVALUATION OF ACUTELY AZOTEMIA PATIENT:

A detailed and accurate history is crucial to aid in diagnosing the type of AKI and determining its subsequent treatment. A detailed history and a physical examination in combination with routine laboratory tests are useful in making a correct diagnosis.

HISTORY

1. History of acute systemic illness such as gastroenteritis, fever with jaundice, (produce AKI by volume depletion, toxic effects).

2. History of chronic systemic illness: A previous illness DM, HT, SLE and chronic symptoms of fatigue, weightloss, anorexia, nocturia and pruritis all suggest chronic kidney disease.

3. Other histories useful to find out etiology are

   a. History of trauma especially in unconscious or comatose patient.

   b. History of drug intake like NSAID, ACE inhibitors, antibiotic like (pencillin, cephalosporin) may cause interstitial nephritis.

   c. History of intoxication – heavy metal compounds, toluene, cuso4, salicylates, ethylene glycol, may explain unexpected episode of AKI.

   d. History of surgery and anaesthesia:

      Both cause vasoconstriction of renal arteries and release of ADH. As a result of reduced volume of urine in early postoperative period,and associated infection with fluid loss leading to prerenal azotemia.

   e. History of unaccustomed exertion – rhabdomyolysis.

PHYSICAL EXAMINATION

PRE RENAL AZOTEMIA is the most common cause of acute kidney injury, adequacy of ECF volume must be carefully determined.

Adequacy of effective blood volume

Body weight, postural blood pressure / pulse change, Skin turgor, mucous membrane moisture.

Recent reduction in body weight, a fall in SBP >10mm Hg or rise in pulse >10/min. tenting of upper thorax skin on pinching, dry mucous membrane all are suggest reduction in ECF volume.

Skin

Examination of skin for petechiae, purpura and ecchymosis provides clues to inflammatory and vascular causes for AKI. Also help to detect DIC hypersensitivity.

Eyes

Evidence of uveitis may indicate interstitial nephritis. Ocular palsy may indicate ethylene glycol poisoning. Findings suggestive of severe Hypertension, endocarditis may be observed after careful examination of eyes.

Look for evidence of intoxication (altered mental status, odour on breath or clothing, muscle tenderness or edema).

Examination of lung, heart, joints, CNS often demonstrate abnormalities.

Abdomen – help to detect obstruction at bladder outlet as the cause of renal failure, may be due to cancer or enlarged prostate. Presence of bruit suggests renal vascular Hypertension.

TESTING PROCEDURES

URINE ANALYSIS

Urine analysis remains the most important test in initial evaluation of AKI.

Prerenal AKI

Urine sediment is characteristically acellular and contains transparent hyaline cast (‘band’ “benign” “inactive” urine sediment). There will be trace or mild proteinuria.

Post renal AKI

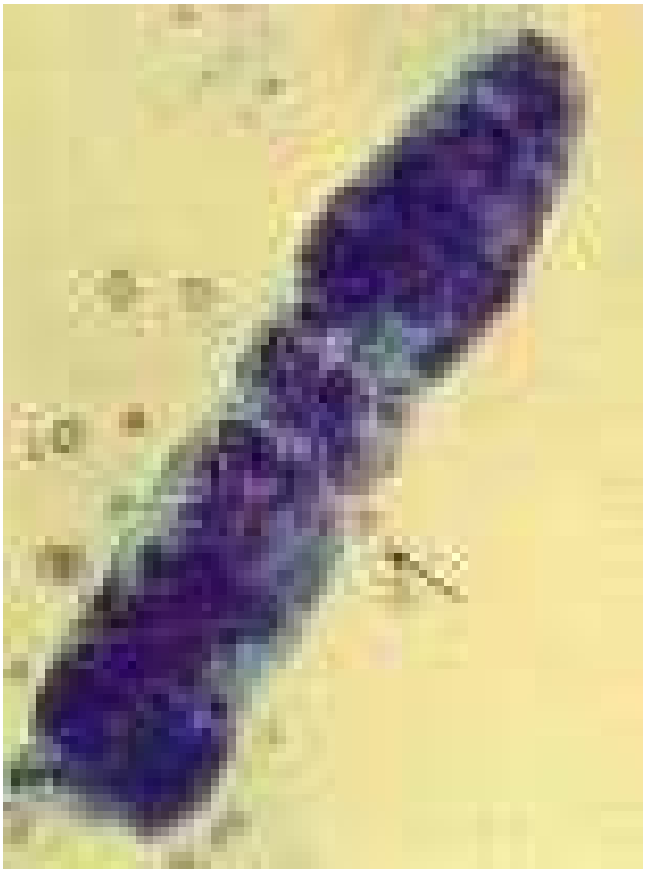
Trace or no proteinuria, can have hemoglobin, or leukocytes.

Intrinsic renal AKI

Intrinsic Renal Azotemia	Proteinuria	Sediments
Ischemic	Mild to moderate proteinuria	Pigmented granular cast
Toxins	Mild to moderate proteinuria	Pigmented granular cast
Acute Interstitial nephritis	Mild-Moderate proteinuria Haemoglobin , leukocytes	White cell and white cell casts, eosinophils and eosinophil casts; redcell.

Occasional uric acid crystals (needle in shape) is common in concentrated urine and also suggest urate nephropathy of prerenal AKI. Oxalate (Envelope shapped), suggest possibility of ethylene glycol ingestion and toxicity.

Muddy brown casts



Red blood cell casts

URINARY DIAGNO STIC INDICES[41]

INDEX	PRE RENAL AKI	RENAL AKI
U_{Na} mEq/l	<10	>20
Urinary Osmolality (mOsmol/kgH_2O)	>500	<250
Urine to plasma urea nitrogen	>8	<3
U_{cr} to P_{cr}	>40	<20
$\frac{\text{Renal failure index } U_{Na}}{U_{cr}/P_{cr}}$	<1	>1
Fractional excretion of Na $\frac{U_{Na} \times P_{cr} \times 100}{P_{Na} \times U_{cr}}$	<1	>1
URINE specific gravity	>1.020	1.010
Plasma BUN / creatinine ratio	>20	<10-15

The FE_{Na} is the most sensitive index. However FE_{Na} may be >1.0% in prerenal AKI if patients are receiving diuretics or have bicarbonaturia (accompanied by sodium to maintain electro neutrality), preexisting chronic kidney disease complicated by salt wasting or adrenal insufficiency. In contrast FE_{Na} is `<1.0% in approximately 15% of patients with nonliguric ischemic or nephrotoxic AKI[29], FE_{Na} is often <1.0 in AKI due to glomerulonephritis, urinary tract obstruction, and vascular disease.

Blood Urea and Nitrogen

Although increased levels of BUN and creatinine are the hall marks of renal failure, the rates of BUN and creatinine increase are very important. Creatinine rises rapidly within 24-48 hours in patients with AKI following renal ischemia, atheroembolization, and radiocontrast exposure. Peak creatinine levels are observed after 3 to 5 days, in contrast nephropathy. The initial rise in serum creatinine is characteristically delayed until the second week of therapy with many tubule epithelial cell toxins (eg aminoglycoside, cisplatin), this reflects the need for accumulation of these agents within cells before GFR falls.

RADIOLOGY

Plain X-ray Abdomen

Less expensive, and easily available. Kidney size, calculi, calcification etc can be diagnosed.

Renal Ultrasound

Useful for
1. Assessing Kidney size.
2. Evaluating obstruction of urinary collecting system
3. Useful for detecting intrinsic renal disease which enhances renal echogenicity, however this finding is nonspecific.
4. Also aids in performing a renal biopsy

Excretory urography

To make out renal size, hydronephrosis, bladder size, bladder outflow tract obstruction.

Ct scan/MRI

Alternative imaging modality to USG. Also useful for evaluation of renal mass, retroperitoneal tumor, renal trauma.

Doppler ultrasound

For assessment of patency of renal artery and vein in suspected vascular obstruction.

Renal angiography

Useful in diagnosis of renal vascular disease, including renal artery stenosis, renal atherothrombotic disease.

Nuclear scan

Radio nucleotide imaging with a Technetium Tc 99m DTPA, 99mTC-DT PA and Iodine 13 Hippurate scan can be used to assess renal renal blood flow and tubular function.

RENAL BIOPSY : INDICATIONS[41]

1. AKI of unknown cause.

2. AKI associated with glomerulonephritis, nephrotic syndrome or vasculitis where acute therapy is contemplated.
3. AKI associated with interstitial disease without a definite cause.
4. Prolonged AKI

NOVEL BIOMARKERS

New biomarkers hold the promise of allowing clinicians to detect kidney injury earlier, to guide future therapy, and to better prognosticate. The currently employed, traditional markers of AKI in the blood (creatinine and urea nitrogen) are insensitive, lagging indicators that are not specific for any given disease process.

Novel biomarkers in Acute Renal Failure[57-59]

Host	Marker	Substrate- test	Comments
Rat	SSAT	Rat kidney- RT-PCR and northern blot	SSAT was able to distinguish AKI with tubular injury from AKI without ATN
Rat & mouse	CYR61	Kidney and urine- western blot	CYR61 is upregulated in kidneys with IRI- able to distinguish prerenal from intrarenal AKI
Human	IL-18	Urine- ELISA	IL-8 elevated in human kidney ATN (native and transplanted kidneys)
Human	NHE3	Urine- semiquantitative immunoblotting	NHE3 differentiated prerenal from intrarenal causes of AKI
Human	Urine proteome pattern	Urine- mass spectroscopy	Humans follwing cardiopulmonary bypass surgery-markers at 2 and 6 hrs postoperatively highly sensitive and predictive of AKI

Human	KIM-1	Urine,kidney-multilple methods	Specific for ischemic AKI/ ATN when compared with other forms of kidney disease
Human, mouse	Gro-α, KC	Urine, blood-ELISA	Gro-α correlates with renal recovery from AKI/DGF in transplant, early increase in urine and blood well before rise in serum creatinine in AKI models
Human	NGAL	Blood, urine-western blot and ELISA	NGAL sensitive, specific and predictive marker of AKI in blood and urine of patients after cardiopulmonary bypass
Human	Actin,IL-6 & IL-8	Urine-dot immunoblot and ELISA	All 3 markers predicted prolonged AKI following renal transplantation in humans

CLINICAL PRESENTATION OF ACUTE KIDNEY INJURY[60]

The clinical feature in patients with AKI may vary depending on the precipitating cause, the severity of renal injury, and the speed with which it develops. In general AKI can present in one of four ways:

1. With an unexpected elevation in BUN and serum creatinine
2. With an alteration in urine flow rate.
3. With the clinical features of the underlying precipitating cause and
4. With clinical or biochemical complications of uremia.

1. AN ASYMPTOMATIC ELEVATION OF BUN OR SERUM CREATININE ON ROUTINE BIOCHEMICAL SCREENING

It is the most common presentation of AKI. Such routine screening has increased recognition of milder cases of AKI which in the absence of oliguria or other complications, might otherwise have gone undetected. However an elevation in BUN or serum creatinine concentration does not always signify a reduction in GFR and other potential causes should be excluded, such as increased protein intake, GI bleed, drug intake (eg. Cephalosporin).

In most cases of AKI, the rate of increase of BUN and creatinine are comparable, ratio of BUN: Creatinine 10 to 20 : 1. A disproportionate increase in BUN may be seen with slow rates of fluid flow along the distal nephron because this enhances renal tubular reabsorption of urea nitrogen. This phenomenon may occur in pre renal condition or obstructive uropathy. In rhabdomyolysis, the serum creatinine levels rises more rapidly, by more than 2 mg/dl/day.

2. ALTERATION IN URINE FLOW RATE

Oliguria – may be the first clue to presence of kidney injury. A urine volume less than 400ml in 24 hours or <20ml/hour is arbitrarily defined as oliguria because this is approximately the minimum volume required to excrete the daily nitrogenous waste products when the urine is maximally concentrated.

Non oliguric failure (Urine output >500ml/day) :- It is common after aminoglycoside nephrotoxicity, burns and administration of radiocontrast dye but it may occur with any cause of AKI. In general non oliguric AKI pretends a better prognosis, possibly because it signifies a lesser degree of renal injury.

Anuria: passage of less than 50ml of urine per day occurs less commonly and indicates a diagnosis of urinary tract obstruction, occasionally seen in RPGN, bilateral cortical necrosis and renal infarction. Alternative Oliguria with Polyuria, which is suggestive of incomplete urinary tract obstruction or occasionally with salt wasting and polyuria.

CLINICAL FEATURES OF THE PRECIPITATING CAUSE OF AKI

AKI has many causes, both extrinsic and intrinsic to kidney. In each case, AKI may present which clinical features related to the underlying cause of renal injury. A thorough history and careful physical examination will be helpful in eliciting these features.

CLINICAL AND BIOCHEMICAL COMPLICATIONS OF AKI

CLINICAL COMPLICATIONS :

1. Neurologic complications

Lethargy and asterixis are neurologic manifestation of uremia that if unrecognized and untreated may progress to confusion, stupor, coma, myoclonus

and seizures. Psychiatric manifestations ranging from severe anxiety to psychosis can occur. These complication can be prevented or treated by dialysis[34].

2. Cardiovascular complications

The most frequent cardiovascular manifestation of AKI is congestive heart failure, which is due to fluid overload. Arrythmias, though common can be due to electrolyte abnormalities[30]. Hypertension is usually due to volume overload and acute glomerulonephritis. Pericarditis occurs with advanced uremia.

3. Hematologic complications

Hematologic complications include anemia, (due to inhibition of erythropoiesis, bleeding, hemodilution, shortened red cell life span)[31] leukocytosis, defective platelet function (due to presence of uremic toxins in plasma), usually improves with dialysis. However, the response to dialysis is often incomplete. Administration of cryoprecipitate or the vasopressin analogue deamino – 8 – d – arginine vasopressin (DDAVP) both improve platelet function in uremia and prolonged bleeding time[32].

4. Gastro intestinal complications

Anorexia, nausea and vomiting are frequent early symptoms. Erosive gastrointestinal ulcers may develop in any part of GI tract, bleeding from these ulcers may be severe and is important cause of death in AKI[33]. Hiccups are a late sign occurring in patients with advanced uremia. Presence of Jaundice is

suggestive of hepatic injury secondary to passive congestion, ischemia or intraabdominal sepsis and is a poor prognostic sign. Mild hyperamylesemia due to reduced renal excretion of amylase.

5. Nutritional and metabolic complications

Hypercatabolism may be releated to uremia per se or is attributable to infection or other acute stress. It has been suggested that elevated parathyroid hormone and metabolic acidosis in AKI may accelerate protein catabolism[35]. Elevated levels of cytokines such as interleukins – 1 and TNF α particularly present in septic patients also contribute to catabolic state. In many patients with AKI, combination of catabolic factors, hormonal imbalance and reduced dietary intake results in negative nitrogen balance. Mild hyperuricemia occurs in AKI, rarely the level exceeds 15 mg/dl.

6. Infectious complications

Between 50 – 90% of patients with AKI develop infections and this high incidence does not appear to be reduced by use of prophylactic antibiotics. The major predisposing factors are: Frequent use of indwelling catheters, and use of peripheral and central intravenous lines for invasive monitoring and as access for hemodialysis. The most frequent sites involved are chest, urinary tract and wounds. The predisposition to serious infection due to defective chemotaxis, absolute or

relative agranulocytosis, relative lymphopenia and impaired cell mediated immunity.

7. Respiratory complications

Pulmonary edema and pneumonia are the common complications. ARDS, seen in critically ill patients with multiorgan failure.

BIOCHEMICAL COMPLICATIONS OF AKI

Abnormalities of fluid and electrolyte metabolism

Fluid retention as a result of decreased salt and water excretion leads to pulmonary edema, cerebral edema, hyponatremia. Fluid overload is common in oliguric patients, surgical patients who receive large quantities of IV fluids perioperatively and in patients requiring fluids for resuscitation. The administration of sodium bicarbonate may be a contributory factor.

Hyponatremia due to excessive administration of hypotonic fluids may be accompanied by lethargy, progressive obtundation and seizures.

Hyperkalemia is one of the serious complications of AKI. In patients with mild AKI, the serum potassium increases by 0.5 to 1.0 meq/l/day, but in patients who are hypercatabolic with extensive tissue injury or hemolysis, the rate of increase is rapid, by 1 to 2 meq/l in only a few hours.

K^+ level 5.5 to 6.5 mEq/L produces tall peaked T waves and shortening of QT interval. With level of 6.5 to 7.5 meq/L QRS complex widend, P wave decreases in amplitude, PR interval is prolonged, ventricular tachycardia is common, and ventricular fibrillations or asystole may be a terminal event[37].

Hypokalemia may be seen rarely in AKI caused by Cisplatin or Amphotericin B induced nephrotoxic non oliguric renal failure.

Metabolic acidosis

In normal person, the metabolism of dietary protein results in daily generation of about 1mEq of fixed, non volatile acids per kg of bodyweight. In AKI, the failure to excrete these acids results in development of metabolic acidosis. In patients with uncomplicated AKI, the serum bicarbonate falls by about 1 to 2 mEq/l/day[38]. In hypercatabolic state, the fall of plasma biocarbonate level is faster.

Abnormalities of divalent cation metabolism[39]

Hypocalcemia is due to retention of phosphate secondary to reduction in GFR, skeletal resistance to calcemic action of parathyroid hormone, and decreased blood level of 1,25 – dihydroxy Vitamin D[40]. Hypocalcemia causes a fall in resting membrane potential with an increase in muscle irritability. It is usually asymptomatic in AKI, possibly because co-existing metabolic acidosis increases the level of serum ionized calcium counter balancing its effect on neuromuscular excitability. Clinically, the symptoms include perioral paresthesias, muscle

twitching, hallucinations, and seizures. Latent tetany may be evident on physical examination. ECG changes include prolonged QT interval.

Hypercalcemia : is due to reduced renal excretion and in some patients by the release of intracellular phosphate. It is seen with severe tissue break down as in rhabdomyolysis and tumour lysis syndrome. Mild degree of asymptomatic hypomagnesemia due to nephrotoxic drugs such as amphotericin B. Hypermagenesemia (due to injudicious use of magnesium containing antacids or laxatives) also seen in AKI.

MANAGEMENT OF ACUTE KIDNEY INJURY

PREVENTION

The initial approach to patients with AKI must be focussed on preventing further injury to the kidney. In many cases this can be achieved by removing the underlying cause and correcting fluid and electrolyte imbalance. There is no specific therapy for ischemic or nephrotoxic AKI. Prevention is important.

1. Careful monitoring of systemic hemodynamics and volume states, especially in elderly and those with preexisting renal insufficiency.

2. Aggressive restoration of intravascular volume reduces the incidence of ischemic AKI after major surgery, burns, trauma or cholera.

3. The incidence of nephrotoxic AKI can be reduced by tailoring the dosage of potential nephrotoxins to body size and GFR.

4. Hypovolemia should be avoided in patients receiving nephrotoxic medications as renal hypoperfusion potentiates the toxicity of most.

Prophylactic use of several regimens to increase renal perfusion (eg. Vasodilators, dopamine, atrial natriuretic peptide) or to maintain high urinary flow (mannitol, loop diuretics) appears to be a logical approach.

SPECIFIC THERAPIES[64]

Prerenal azotemia is rapidly reversible on restoration of renal perfusion. The composition of replacement fluids for treatment of hypovolemia varies depending on the source of fluid loss. Hypovolemia caused by hemorrhage is ideally corrected with packed red cells if the patient is hemodynamically unstable or if the hematocrit is low. Isotonic saline is usually appropriate replacement for mild to moderate hemorrhage or plasma loss (e.g burns, pancreatitis). Urinary and gastrointestinal fluids are hypotonic. Initial replacement is by hypotonic solutions (eg. 0.45% saline), and subsequent therapy should be on measurements of volume and ionic content of excreted or drained fluids. Cardiac failure may require aggressive management with positive inotropes, preload and after load reducing agents, antiarrhythmic drugs, and mechanical aids such as intra aortic balloon pumps.

Fluid management is challenging in patients with AKI and cirrhosis. The relative contribution of hypovolemia to AKI is determined only by administration of fluid challenge, often with invasive monitoring of systemic hemodynamics. Fluids should be administered slowly, as non responders may suffer an increase in ascites formation and/or pulmonary edema. Large volume paracentesis will improve GFR, possibly by lowering intra abdominal pressure and promoting blood flow in renal veins.

INTRINSIC AKI

A variety of agents have been tested for their ability to attenuate injury or hasten recovery in ischemic and nephrotoxic ATN. However, none was consistently been shown to benefit. These include strategies to increase renal blood flow and urine flow (e.g. lowdose dopamine, atrial natriuretic peptide, mannitol, loop diuretics), relieve tubular obstruction (e.g RGD peptide), reduce epithelial cell swelling (eg mannitol), replenish cell ATP level (Mg ATP), scavenge oxygen free radical, (e.g superoxide dismutase, catalase, mannitol), inhibit leukocyte endothelial cell adhesion during reperfusion (e.g anti-CD18, anti ICAM-1 or anti-p-selectin monoclonal antibodies) stimulate cellular regeneration (e.g. epidermal growth factor, aminoacid infusion) and prevent accumulation of intracellular calcium (e.g. calcium channel blocker).

AKI caused by other intrinsic renal disease such as acute glomerulonephritis or vasculitis may respond to corticosteroids, alkaylating agents and/or plasmapheresis, depending on primary disease. Aggressive control of systemic arterial pressure is of paramount importance in limiting renal injury in malignant hypertensive nephrosclerosis, toxemia of pregnancy and other vascular diseases. Hypertension and AKI associated with scleroderma may be exquisitely sensitive to treatment with ACE inhibitors. Antiplatelet agents, plasma exchange, and plasma infusion are useful in treatment of HUS and TTP.

POST RENAL AKI

Obstruction of urinary tract should be treated by appropriate surgical procedure. Eg. Removal of calculus.

SUPPORTIVE MEASURES

DIET AND FLUIDS

1. Fluid intake should be restricted to 500 ml plus measured losses.
2. Salt (1-2g/d), potassium intake (<40mmol/d), and phosphate intake (<800mg/d) is essential.
3. Calorie intake of at least 2000 kcl/day restricts the catabolism of protein for energy and slows the formation of nitrogenous metabolites.

HYPERKALEMIA

If untreated may lead to ventricular tachycardia and death. Restrict dietary K^+ intake, eliminate K^+ supplements, K^+ sparing diuretics Potassium binding ion exchange resins, (eg, sodium polystyrene sulphonate) administrated orally or rectally, can bind potassium in the gut. Extracellular potassium concentrations can be temporarily lowered by administering glucose and insulin. Calcium gluconate (10ml of 10% solution over 5 min intravenous) will buffer the effects of potassium on myocardium. Dialysis is indicated if hyperkalemia is resistant to these measures.

MANAGENT OF ISCHEMIC AND NEPHROTOXIC ACUTE KIDNEY INJURY[64]

Management Issue	**Therapy**
Reversal of Renal Insult	
Ischemic ATN	Restore systemic hemodynamics and renal perfusion through volume resuscitation and use vasopressors.
Nephrotoxic ATN	Eliminate nephrotoxic agents Consider toxin-specific measures: e.g., forced alkaline dieresis for rhabdomyolysis, allopurinol/ rasburicase for tumor lysis syndrome.

Prevention and Treatment of Complications

Intravascular volume overload	Salt and water restriction, Diuretics, Ultrafiltration
Hyponatremia	Restriction of enteral free water intake, Avoidance of hypotonic intravenous solutions, including dextrose-containing solutions.
Hyperkalemia	Restriction of dietary K^+ intake, Eliminate K^+ supplements and K^+ sparing diuretics, Loop diuretics to promote K^+excretion, Pottassium binding ion-exchange resins (e.g., sodium polystyrene sulfonate or Kayexelate), Insulin (10 units regular) and glucose (50 mL of 50% dextrose) to promote intracellular mobilization, inhaled β-agonist therapy to promote intracellular mobilization. Calcium gluconate or calcium chloride (1g) to stabilize the myocardium, Dialysis.
Metabolic acidosis	Sodium bicarbonate (maintain serum bicarbonate >15 mmol/L or arterial pH > 7.2)

	Administration of other bases, e.g., THAM ,Dialysis.
Hyperphosphatemia	Restriction of dietary phosphate intake, Phosphate binding agents (calcium carbonate, calcium acetate, sevelamer hydrochloride, aluminum hydroxide).
Hypocalcemia	Calcium carbonate or gluconate (if symptomatic)
Hypermagnesemia	Discontinue Mg^{++} containing antacids.
Hyperuricemia	Treatment usually not necessary if <890 μmol/L or <15mg/dL.
	Allopurinol, forced alkaline dieresis, rasburicase.
Nutrition	Protein and calorie intake to avoid net negative nitrogen balance.
Dialysis	To prevent complications of acute kidney injury.
Choice of agents	Avoid other nephrotoxins: ACE inhibitos/ARBs, aminoglycosides, NSAIDs, radiocontrast unless absolutely necessary and no alternative.
Drug dosing	Adjust doses and frequency of administration for degree of renal impairment.

RENAL REPLACEMENT THERAPY : DIALYSIS

Dialysis replaces renal function until regeneration and repair, restore renal function Indications[41] are

1. Symptomatic uremia

   (asterixis, encephalopathy etc),
2. Acidosis- refractory to medical treatment
3. Hyperkalemia- refractory to medical treatment
4. Volume overload- refractory to medical treatment

Types of dialysis available are peritoneal, hemodialysis or hemofiltration. Dialysis modality is choosen according to the needs of individual patients (e.g., peritoneal dialysis may be preferable if the patient is hemodynamically unstable, haemodialysis after abdominal surgery involving peritoneum), the expertise of the nephrologist and the facilities of institution.

PERIOTONEAL DIALYSIS

Is simple, safe, relatively "Low tech" and portable[42]. An intact abdomen is essential for efficient function. It is achieved by insertion of a single lumen cuffed catheter into peritoneal cavity. Sterile electrolyte (dialysate) solution is infused into the abdomen and allowed to dwell for a short period. Metabolites diffuse from the capillaries through the peritoneal membrane while the fluid is in peritoneal cavity. Repeated exchanges will slowly remove urea, creatinine, Potassium etc. A high

concentration of glucose in the dialysate acts as an osmotic agent. It is less efficient than hemodialysis.

Benefits of peritoneal dialysis include avoidance of systemic anticoagulation and need for angioaccess. Systemic hypotension is typically avoided. Drawbacks include the risk of visceral injury during catheter placement and peritonitis subsequently. With the development of Intermittent hemodialysis and more recently the slow continuous blood purification therapies there has been decline in the use of peritoneal dialysis in acute setting[43.]

HEMODIALYSIS

Is more efficient than peritoneal dialysis. Vascular access for intermittent hemodialysis is best achieved by insertion of a temporary double-lumen hemodialysis catheter into the internal jugular vein. The subclavian and femoral veins are alternative access sites. Anticoagulation with heparin is standard method of preventing thrombosis of the extra corporeal circuit during acute intermittent dialysis[45]. Hemodialysis uses a synthetic membrane in the artificial kidney or dialyser. The choice of membrane used during dialysis may have an effect on outcome. Several randomized controlled trials indicate maintenance phase of ATN is significantly shorter with use of more biocompatible synthetic dialysis membrane than with conventional cuprophane or cellulose acetate membranes probably because of less activation of blood complement, leukocytes and other mediator

system[44]. By ultrafiltration excess body fluid can be removed by placing a pressure difference across membrane. The machine manufactures dialysate from purified water and a concentrated electrolyte solution, checks its composition and temperature and passes it on to the dialyses.

Complication of acute intermittent hemo dialysis

1. Hypotension
2. Muscle cramps
3. Dialysis disequilibrium syndrome : Typically occurs after a first dialysis in very uremic patients. It is self limited, characterized by nausea, vomiting, headache, altered consciousness and rarely seizures or coma. It is due to rapid movement of water into brain cells following development of transient plasma hypo-osmolality as solutes are rapidly cleared from bloodstream during dialysis.
4. Anaphylactoid reactions to dialyzer on its first use, rarely seen[46]

For those who do not tolerate intermittent hemodialysis, and peritoneal dialysis, continuous arteriovenous hemodiafiltration (CAVH) and continuous venoveous hemodiafiltration (CVVH) are alternative techniques for treatment of AKI.

PROGNOSIS OF AKI

The mortality rate among patients with AKI approximates 50% and has changed little over the past 30 years despite of advent of improved nutritional therapy, refined dialysis technique, advances in antibiotic therapy. This may be explained by the two demographic changes: the age of the patients continues to rise, and co-existing serious illnesses are increasing among patients[47]. It should be stressed, however that patients usually die from sequalae of the primary illness that induced AKI and not from AKI.

The mortality rates vary greatly depending on the cause of AKI: 15% in obstetric patient, 30% in toxin related AKI and 60% following trauma or major surgery. If the renal failure is mild, the mortality rate is 30-60% and if the patient need dialytic therapy, the mortality rate is 50-90%. Mathematical models for outcome of AKI in critically ill patients (Apache II, Billock, cioffi) are not reliably predictive. However death is almost universal if AKI is associated with failure of more than three other organ systems[48].

Non oliguric AKI carries better prognosis than oliguric AKI, partly because management of fluid and electrolyte abnormalities is easier and these patients have a less severe renal insult. The influence of age as independent risk factor for mortality is not fully defined. Most patients who survive an episode of AKI recover sufficient renal function to live normal lives.

PROGNOSTIC FACTORS IN AKI

General	Controversial
Hypotension Sepsis Oliguria Need for assisted ventilation Severity of renal failure Failure of other organs Nature of underlying illness	Age over 65 years Magnitude of rise in serum creatinine

Early diagnosis, prevention, and treatment of sepsis, prevention of complications of uremia are the major goals of management in AKI patients[49].

OUTCOMES

The crude mortality rate among patients with intrinsic AKI approximates 50% and has changed little over the past 3 decades. The lack of improvement in outcome, despite significant advances in supportive care may be more apparent than real and reflect a reduction in the percentage of isolated AKI combined with an increase in AKI complicating the multi organ dysfunction syndrome. When allied with the current trend for more aggressive surgical and medical intervention in ageing population. These factors probably mask an improvement in outcome. Mortality rates differ markedly depending on the cause of AKI: being approximately 15% in obstetric patients, 30% in toxin related AKI, and 60% to 90% in patients with sepsis[60].

MATERIALS AND METHODS

Place	:	Department of Medicine
Design	:	Observational Study
Period	:	September 2007 to September 2008
Sample Size	:	100 Patients
Collaborating Departments	:	Nephrology

Inclusion Criteria

An absolute increase in serum creatinine of more than or equal to 0.3 mg/dl (≥ 26.4 μmol/l), a percentage increase in serum creatinine of more than or equal to 50% (1.5-fold from baseline)

Exclusion Criteria

1. Patients with chronic kidney disease
2. Patients with abnormal kidney size and abnormal corticomedullry

Differentiation

A thorough diagnostic evaluation was made by a detailed history, physical examination, urine analysis, complete hemogram, renal function tests, renal

ultrasound in appropriate places, serology for leptospirosis, enteric fever, and smear for malaria and other relevant investigations to find out the etiological diagnosis. The proforma used for the same is attached.

Once the diagnosis was made, the patients were started on appropriate therapy. Wherever possible the etiological factors were treated or the offending agents withdrawn. Renal replacement therapy was given according to the standard clinical and biochemical indications.

Ethical Committee approval was obtained.

OBSERVATIONS & DATA ANALYSIS

Total Number of Patients –100

Male	57
Female	43
Age group range	13-85 yrs
Mean age	41.4

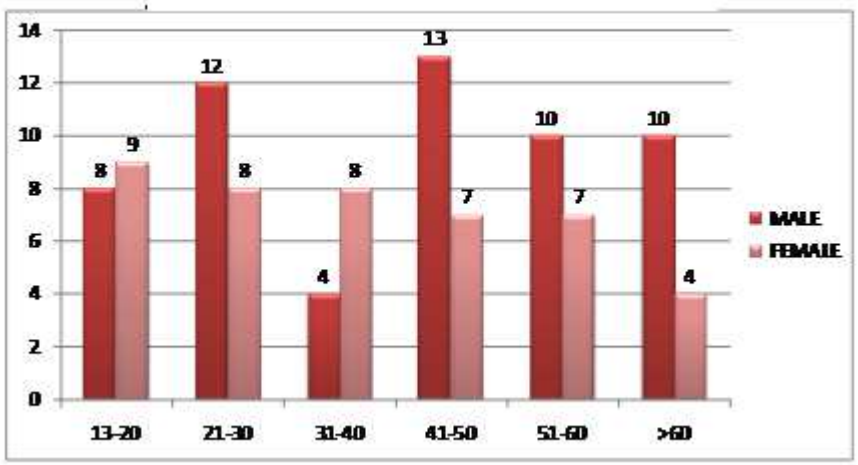
The most common clinical features observed in our study were oliguria (88%), vomiting (64%), facial puffiness (53), loose stools (51%), pedal oedema (47%), altered sensorium (13%), jaundice (11%).

TABLE – 1

AGE DISTRIBUTION ACUTE KIDNEY INJURY IN OUR STUDY

AGE(Years)	MALE	FEMALE	TOTAL (n=100)
13 – 20	8	9	17
21 – 30	12	8	20
31 – 40	4	8	12
41 – 50	13	7	20
51 – 60	10	7	17
> 60	10	4	14

AGE DISTRIBUTION OF ACUTE KIDNEY INJURY



In our study more cases occurred in second & fourth decades. In our study we got about 14 cases in >60 yrs. In that about 10 patients were included in risk, 1 patient in injury & 3 patients in failure based on RIFLE criteria. Two patients were treated with hemodialysis & remaining cases treated conservatively. Two cases were died of complications during the management mainly due to cardiovascular complications.

TABLE - 2

PRESENTING FEATURES OF AKI IN OUR STUDY

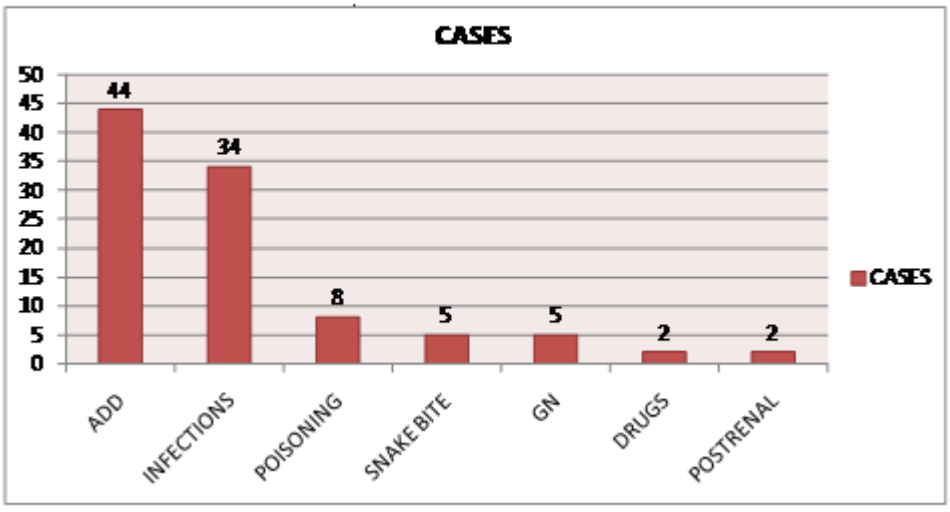
URINE OUTPUT	Male	Female	Total (n – 100)
OLIGURIC	50	38	88
NONOILGURIC	7	5	12

TABLE – 3

ETIOLOGICAL PROFILE AKI IN OUR STUDY

ETIOLOGY	Males	Females	Total(n=100)
ADD	23	21	44
SEPSIS	6	4	10
MALARIA	5	3	8
LEPTOSPIROSIS	2	2	4
LEPTO+ MALARIA+ ENTERICFEVER	8	4	12
POISONING	6	2	8
GLOMERULO NEPHRITIS	0	5	5
SNAKE BITE	2	3	5
DRUG INDUCED	1	1	2
POST RENAL (BPH, CALCULUS)	2	0	2

ETIOLOGICAL PROFILE OF AKI



In our study, maximum cases of AKI were due to acute diarrheal diseases (44%). The next one was infections (34%) like malaria (including complicated), leptospirosis, enteric fever and combination of above infections. Poisoning(8%) occupies the next position(includes cuso4 poison, dichromate poison, paracetamol poison, & unknown poison). My study also contain glemerulonephritis (5%), snake bite (5%), drugs (2%-ACEI, NSAIDS), postrenal(2%) as causes of remaining AKI.

TABLE – 4

CAUSES OF NONOLIGURIC AKI IN OUR STUDY

CAUSES	CASES
ADD	3
POISONING	3
DRUG INDUCED	2
SNAKE BITE	2
SEPSIS	1
MALARIA	1

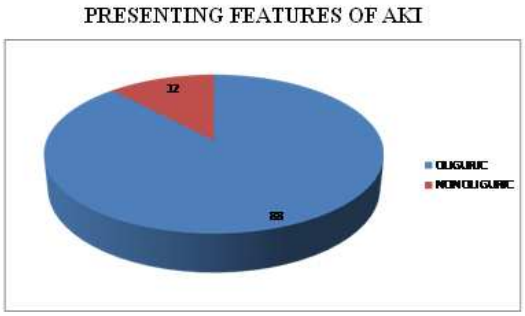


TABLE – 5

ETIOLOGY OF ACUTE KIDNEY INJURY IN OUR STUDY

ETIOLOGY	NO OF CASES	PRERENAL	RENAL	POSTRENAL
ADD	44	40	4	0
SEPSIS	10	1	9	0
MALARIA	8	5	3	0
LEPTOSPIROSIS	4	4	0	0
LEPTO+ MALARIA+ ENTERICFEVER	12	10	2	0
POISONING	8	3	5	0
GLOMERULO NEPHRITIS	5	0	5	0
SNAKE BITE	5	0	5	0
DRUG INDUCED	2	1	1	0
POST RENAL (BPH, CALCULUS)	2	0	0	2

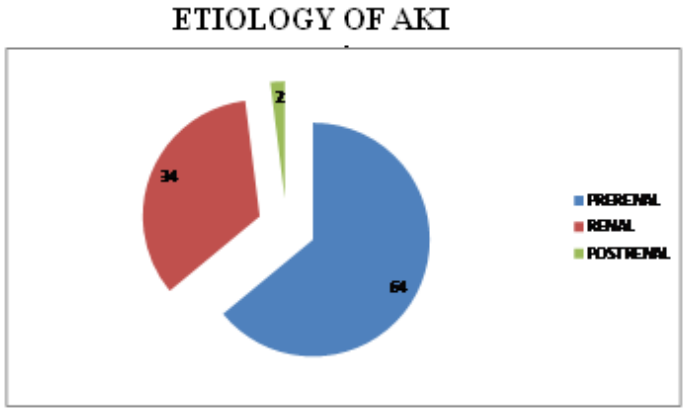


TABLE – 6

AKI- RIFLE DITRIBUTION IN OUR STUDY

ETIOLOGY	NO OF CASES	RISK	INJURY	FAILURE
ADD	44	28	10	6
SEPSIS	10	0	0	10
MALARIA	8	2	2	4
LEPTOSPIROSIS	4	3	1	0
LEPTO+ MALARIA+ ENTERICFEVER	12	10	1	1
POISONING	8	1	4	3
GLOMERULO NEPHRITIS	5	0	5	0
SNAKE BITE	5	1	1	3
DRUG INDUCED	2	1	1	0
POST RENAL (BPH, CALCULUS)	2	0	1	1

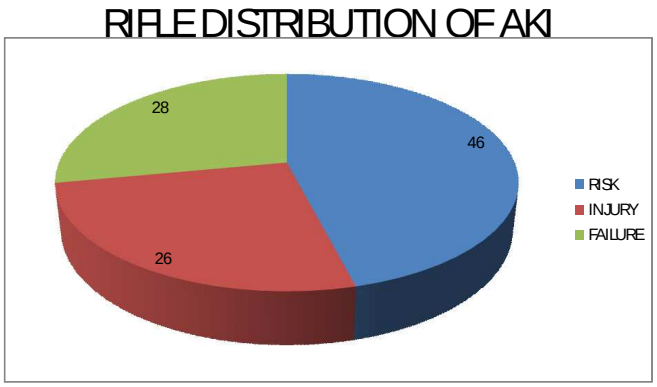
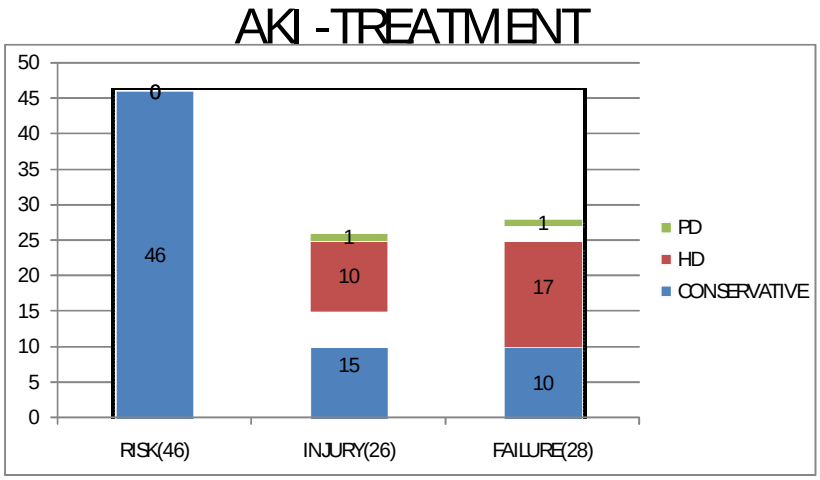


TABLE – 7

ACUTE KIDNEY INJURY-TREATMENT

	NO OF CASES	CONSERVATIVE	HD	PD
RISK	46	46	0	0
INJURY	26	15	10	1
FAILURE	28	10	17	1



In our study based on RIFLE criteria 46 patients were included in risk, 26 in injury, 28 in failure. In risk all patients were treated conservatively & all patients survived. In injury 57.6% of patients were treated conservatively, 42.3% of patients were dialysed & one patient died. In failure 35.7% of patients were treated conservatively, 64.2% of patients were treated with dialysis and 12 patients died.

The frequency of peritoneal dialysis is decreased so drastically because of availability of Hemodialysis and higher efficiency of hemodialysis. In our study about 26% of patients had complications and 13 patients (50%) were treated successfully.

TABLE – 8

ACUTE KIDNEY INJURY-OUTCOME

	NO OF CASES	SURVIVED	EXPIRED
RISK	46	46	0
INJURY	26	25	1
FAILURE	28	16	12

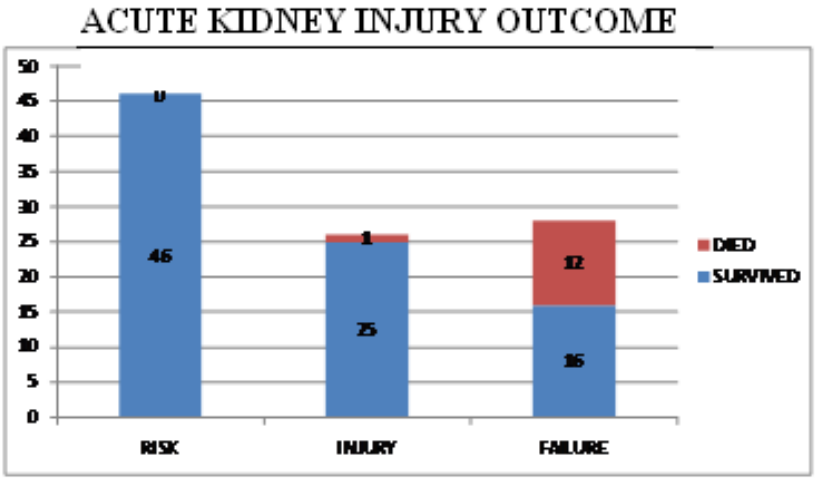
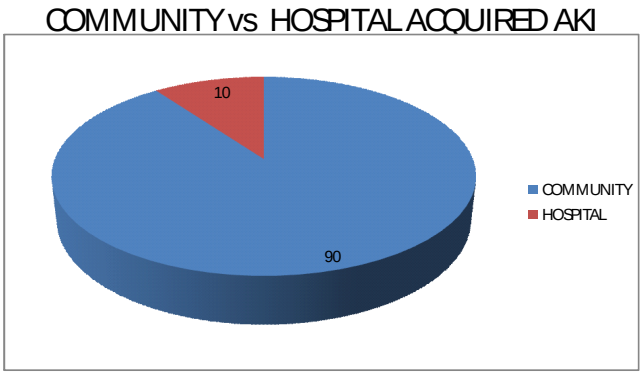


TABLE – 10

HOSPITAL ACQUIRED AKI

ETIOLOGY	CASES
SEPSIS	8
DRUGS	2



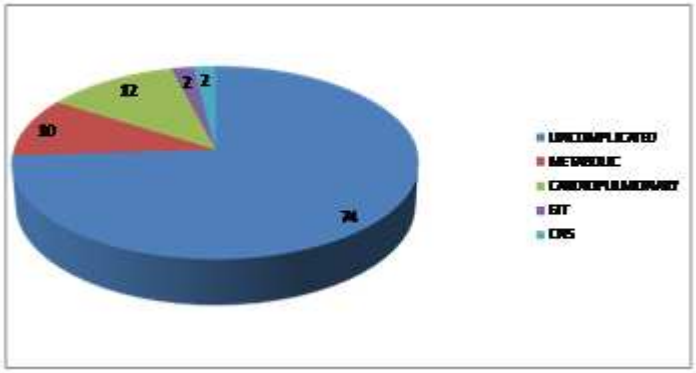
In our study about 10 patients were hospital acquired AKI. In that 4(40%) patients were treated with hemodialysis, 6(60%) patients were treated conservatively, In that 5 (50%) of patients were treated successfully.

TABLE – 9

ACUTE KIDNEY INJURY-COMPLICATIONS

COMPLICATIONS	CASES
METABOLIC	10
CARDIOPULMONARY	12
GIT	2
CNS	2
TOTAL	26

COMPLICATED AKI



DISCUSSION

Acute kidney injury is a potentially fatal, but reversible renal disease. The etiology, course , outcome, differ in various parts of india due to its climatic & geographic diversity & the varying standards of medical care.

In our study 100 patients were analysed , there were 57 males & 43 females. The median age was 41.4. . Maximum number of cases occurred in the second & fourth decade .

In our study oliguric AKI predominate (88%). This is in concordance with a previous study by **Dr.M.A.Muthusethupathi et al [67]** in 1991.

Acute diarrhoeal disease was the leading cause of AKI in our study. **Cengiz utas et al[66]** from turkey reported diarrhoeal disease was the commonest cause of AKI. **Dr.M.A.Muthusethupathi et al[67]** reported that leptospirosis was the leading cause of AKI in chennai.

Acute diarrhoeal disease was the leading cause of AKI in our study. Of 44(44%) patients 5 (11.3%) patients were treated with dialysis (Peritoneal Dialysis=2, Hemodialysis =3), 39(88.6%) cases treated conservatively. One patient died due to arrythmia . The mortality observed during the study was 2.2%, which is less when compared with the reports of a study by **Dr.M.A.Muthusethupathi et al [67]** (34.7%) . **SK Agarwal et al[65]** in their study reported 11% cases of AKI was

due to diarrhoeal dieases in north India . Awareness of early rehydration therapy and early referral to higher centers contribute to decline in mortality.

Malarial AKI was observed in 8(8%) cases. In **Dr.M.A.Muthusethupathi et al** [67] study no cases were reported. A study by **Prakash J et al**[68] from eastern india reported 4.2 % cases were reported. Out of 8 cases 4 cases were treated with hemodialysis and 4 cases were treated conservatively. The mortality observed in our study was 25% in contrast with 42.5% in **Zinna S et al**[36]**.** Two patients presented with cerebral malaria with renal failure. One patient had metabolic acidosis, One patient had arrhythmias, One patient had hypotension. Mortality decreased mainly due to earlier diagnosis and earlier use of hemodialysis.

We have 4 (4%) patients 0f leptospirosis with AKI as compared to **Dr.M.A.Muthusethupathi et al**[67] study in which the incidence of leptospirosis was 41% . we confirmed the cases based on modified faine's criteria. Of 4 patients 3 patients were treated conservatively, one patient was treated with hemodialysis. There was no mortality in our study . In the study by **Dr.M.A.Muthusethupathi et al**[67] the mortality was 20.8%. The low mortality is mainly due to awareness of leptospirosis, its standard diagnostic criteria(modified faine's criteria), therapy and early referral to higher center .

In contrast with other studies our study had significant number of patients with the combination of leptospirosis, malaria, enteric fever. In other studies, no

such combinations were reported. Of 12(12%) patients, 10 (80%) were treated conservatively, two patients were treated with hemodialysis. We also had significant number of sepsis (10%) as a cause of AKI. Of 10 patients 4(40%) patients were treated conservatively, 6(60%) patients were treated with hemodialysis. The mortality was higher (70%). It may be due to association with comorbid illnesses (eg. Diabetes), and higher incidence of multi organ dysfunction syndrome in these patients.

AKI due to cupper sulphate poisoning contributes about 3(3%) . All 3 patients were treated with Hemodialysis and recovered. **In Dr.M.A.Muthusethupathi et al[67]** study the incidence was 3.4% and the mortality was 37.5%. In **Prakash J et al[68]** study no cases were reported. All 3 patients in our study came under the 'Injury 'criteria. One patient developed metabolic acidosis and was, treated with dialysis successfully. Another 2 patients developed AKI after dichromate poisoning, Both patients were treated with hemodialysis . Both patients developed metabolic acidosis and one patient died. Another 2 patients developed AKI after unknown poisoning, one patient was treated with hemodialysis. Both patients developed metabolic acidosis and one patient died. One patient developed AKI after paracetamol poisoning and was treated with hemodialysis. There was not many studies on dichromate and paracetamol poisoning with acute kidney injury.

The 5 patients had snake bite as a cause of AKI. 4(80%) patients were treated with hemodialysis. The mortality Was 20%. In **Dr.M.A.Muthusethupathi**

et al[67] study the incidence was 4.7% and the mortality was 34.7%. In **Prakash J et al** [68] study no case of snake bite was reported. In our series mortality is mainly due to delay in seeking early treatment as most of the patients were from rural areas.

In our study, 5(5%) patients had glomerulonephritis. All patients were treated conservatively and recovered. AKI due to glomerulonephritis was 8.5% in **Dr.M.A.Muthusethupathi et al** study .

AKI due to drugs contribute 2(2%) of patients. One patient was hypertensive and developed acute kidney injury, when he was started on ACEI, however his renal Doppler was normal, and he recoverd. One patient was diagnosed to have rheumatoid arthritis, and developed acute kidney injury with NSAIDS and recovered with conservative management. In **Prakash J et al**[68] study the incidence was 2% which was similar to our study.

AKI due to Postrenal (surgical) causes contribute 2(2%) of patients. One patient had bilateral l staghorn calculi treated surgically, and he recovered. One patient had BPH of grade 3 and was treated surgically. He recovered. This is similar to a previous study by **Dr.M.A.Muthusethupathi et al** [67] in 1991(2.5% incidence).

Of total 100 cases 46 patients came under RISK based on “RIFLE” criteria. In that 100% patients were treated successfully with conservative management. The

mortality of RISK in our study was nil. About 26 patients came under INJUR. In that 15 (57.6%) patients treated conservatively, 11(42.3%) patients were treated with dialysis. The mortality in INJURY was 3.8%. About 28 patients came under FAILURE. In that 10 (35.7%) patients were treated conservatively, 18 (64.2%) patients were treated with dialysis. The mortality in FAILURE was 46.1%. **Ostermann M, Chang RW: CCM 2007**[63] detected the following RIFLE class F have a mortality of 57%, RIFLE class I 45% RIFLE class R 21%. There was an association between acute kidney injury and hospital outcome, but associated organ failure, had a greater impact on prognosis than severity of acute kidney injury.

CONCLUSION

The presentation of AKI is predominatly oliguric . But nonoliguric AKI should be born in mind.

Acute diarrhoeal disease still remains the most common cause of acute kidney injury. Mortality observed during the study is very low when compared the previous studies.Awareness of early rehydration therapy and early referral to higher centers contribute to decline in mortality.

Infections occupies significant place in the etiology of AKI, which includes malaria (including complicated), leptospirosis, enteric fever, and combination of above infections. Regarding sepsis we have to prevent the multi organ failure or we have to treat the multi organ failure very aggressively to reduce the higher mortality associated with sepsis.

Majority of our patients were managed conservatively

When dialysis is indicated for clinical or biochemical reasons,

HD is the preferred mode of dialytic support. PD is begun only when HD is

Not available or is contraindicated

Main issues obtained were,

1. About two-third of patients with AKI can be managed by conservative measures alone.
2. About one-third of patients needed dialysis,

Delayed diagnosis and treatment, pulmonary and other infections, the frequent presence of complicating features and the multi organ dysfunction syndrome are the chief reasons for the mortality rate in our study.

The frequency of peritoneal dialysis is decreased so drasticaly because of availability of hemidialysis and higher efficiency of hemodialysis.

The RIFLE classification was suitable for the definition of acute kidney injury in all type of medical settings. There was an association between acute kidney injury and hospital outcome, but associated organ failure, had a greater impact on prognosis than severity of acute kidney injury.

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CLINICAL PROFILE OF ACUTE KIDNEY INJURY DUE TO MEDICAL DISORDERS IN CHENNAI

NAME: AGE:

SEX : OCCUPATION :

IP NO :

PRESENT ILLNESS

OLIGURIA Y/ N

PEDAL OEDEMA Y/N

FACIAL PUFFINESS Y/N

HAEMATURIA Y/N

THIRST Y/N

ORTHOSTATIC DIZZINESS Y/N

LOOSE STOOLS Y/N

VOMITING Y/N

GI HEMORRHAGE Y/N

BURNS Y/N

TRAUMA Y/N

BREATHLESSNESS Y/N

NYHA CLASS

1. PND/ORTHOPNOEA

ASCITIS Y/N

DRUG INTAKE Y/N

DRUG NAME:

DURATION :

ABDOMINAL PAIN Y/N

IF YES 1) FLANK PAIN/ SUPRAPUBIC

2) RADIATION Y/N

FEVER Y/N

IF YES DESCRIBE

PYURIA Y/N

RASH Y/N

JAUNDICE Y/N

INSECT BITE/SNAKE BITE Y/N

POISONING Y/N

NOCTURIA Y/N

URGENCY Y/N

FREQUENCY Y/N

HESITANCY Y/N

POLYURIA Y/N

BOWEL INCONTINENCE Y/N

IMPOTENCE Y/N

PAST HISTORY

DIABETES Y/N

CAHD Y/N

HYPERTENSION Y/N

PERIPHERAL VASCULAR DISEASE Y/N

CARDIAC FAILURE Y/N

LIVER FAILURE Y/N

RENL DISEASE Y/N

MAJOR SURGERY Y/N

UTI Y/N

PERSONAL HISTORY

SMOKING	Y/N
ALCOHOL	Y/N
NSAIDS USE	Y/N
DRUG ABUSE	Y/N

FAMILY HISTORY

DIABETES	Y/N
HYPERTENSION	Y/N
CAHD	Y/N
RENAL DISEASE	Y/N

EXAMINATION

CONCIOUSNESS	NORMAL/ALTERTED
PALLOR	Y/N
CYANOSIS	Y/N
CLUBBING	Y/N
PEDAL EDEMA	Y/N
FACIAL PUFFINESS	Y/N
JVP	NORMAL/ELEVATED
TEMPRATURE	
BLOOD PRESSURE	
PULSE RATE	
URINE OUTPUT	

CARDIOVASCULAR SYSTEM

HEART SOUNDS	
MURMUR	Y/N
S3\S4\RUB	Y/N

RESPIRATORY SYSTEM

RALES Y/N

PLEURAL EFFUSION Y/N

GASTERO INTERSTINAL SYSTEM

TENDERNESS Y/N

IF YES SPECIFY THE AREA:

FREE FLUID Y/N

ORGANOMEGALY Y/N

IF YES DESCRIBE:

CENTRAL NERVOUS SYSTEM

CONCIOUSNESS NORMAL/ALTERED

ORIENTATION NORMAL/ ALTERED

ASTERXIS Y/N

INVESTIGATIONS

1) RENAL FUNCTION TESTS

SUGAR						
UREA						
CREATININE						
SODIUM						
POTTASIUM						
BICARBONATE						
PH						

2) HEMOGRAM

HB :

TC :

DC :

ESR :

PLATELETS :

3) URINE

AIBUMIN :

SUGAR :

DEPOSITS :

RBCS :

CASTS :

HB :

SODIUM :

CREATININE :

4) URINE PROTEIN

CREATININE RATIO :

5) FE NA :

6) ECG :

7) CHEST X-RAY :

8) USG ABDOMEN :

9) MSAT/MAT :

10) PERIPHERAL

SMEAR/QBC :

11) BLOOD C/S :

12) URINE C/S :

13) LFT :

FINAL DIAGNOSIS

RIFLE CRITERIA RISK / INJURY / FAILURE

TYPE OF AKI PRERENAL/RENAL/ POSTRENAL

ETIOLOGY

COMPLICATIONS

TREATED : CONSERVATIVELY\HEMODIALYSIS/ PERITONEAL DIALYSIS

OUTCOME : RECOVERED\DIED

HOSPITAL ACQUIRED / COMMUNITY ACQUIRED

S.no	Age	Sex	Ip no	Oliguria	Fever	Dirrhoea	Vomiting	PE	Abd pain	Poisoning	Snake bite	DM	HTN	BP	Fever	Jaundice	PE	Facial puffiness	Breathlessness	Altered sensorium	urine alb	PCR
1	48	M	19937/07	1	1	2	2	1	2	2	2	2	2	140/90	1	2	1	1	2	2	2+	
2	80	M	21228/07	1	2	1	1	2	1	2	2	1	2	160/80	2	2	1	1	2	2	3+	
3	48	F	21919/07	1	1	1	1	1	2	2	2	2	1	100/70	1	2	1	2	1	2	2+	0.29
4	25	M	19361/07	2	2	2	1	2	1	1	2	2	2	169/100	2	2	2	2	1	1	2+	1.3
5	20	F	25813/07	1	2	2	1	2	1	1	2	2	1	140/100	2	2	2	1	1	2	1+	
6	40	F	11221/07	1	2	1	1	2	2	2	2	2	2	110/70	2	2	1	1	2	2	3+	
7	17	M	20241/07	2	1	2	2	2	2	2	2	2	2	130/80	1	1	2	1	2	2	2+	0.6
8	20	F	28219/07	1	2	1	1	1	1	2	2	2	2	170/110	2	2	1	1	2	2	3+	
9	27	F	11976/07	1	1	2	2	2	2	2	2	2	2	110/80	1	2	2	2	2	2	2+	
10	60	M	19337/07	1	1	2	2	2	2	2	2	1	1	140/90	1	1	1	1	2	2	1+	
11	29	M	22928/07	1	1	2	1	2	2	2	2	1	2	110/80	1	2	1	1	1	2	3+	
12	17	F	27850/07	2	2	2	2	2	2	2	1	2	2	120/80	2	2	2	2	2	2	nil	
13	36	M	22937/07	1	2	1	1	2	2	2	2	2	2	150/90	2	2	2	2	2	2	2+	
14	17	F	21187/07	1	1	2	1	2	2	2	2	1	2	160/90	1	2	1	1	1	2	3+	1.4
15	30	M	23621/07	1	2	1	1	2	2	2	2	2	2	130/80	2	2	2	2	2	2	3+	
16	75	M	23418/07	1	2	1	2	2	1	2	2	2	2	150/100	2	2	2	2	2	2	3+	
17	45	F	21136/07	1	2	1	2	1	1	2	2	1	2	120/70	2	2	1	2	2	2	2+	0.4
18	18	M	22791/07	1	2	1	1	1	1	2	2	1	2	130/100	2	2	1	1	2	2	2+	1.5
19	40	F	28437/07	1	1	2	1	1	1	2	2	2	1	130/70	1	2	1	1	1	2	3+	
20	55	M	18189/07	1	2	2	2	2	1	2	2	2	2	156/80	2	2	2	2	2	2	1+	
21	43	F	29919/07	1	2	1	1	1	2	2	2	2	1	180/90	2	2	1	1	2	2	2+	0.5
22	13	M	21038/07	2	2	2	2	2	2	1	2	2	2	110/90	2	2	2	1	1	2	3+	
23	60	M	23661/07	1	1	1	1	1	2	2	2	1	2	150/80	1	1	1	1	1	2	1+	2
24	45	M	22932/07	1	2	1	1	2	2	2	2	2	2	140/80	2	2	2	2	2	2	3+	
25	35	F	34098/07	1	1	1	1	2	2	2	2	2	2	130/90	1	2	1	2	2	1	3+	
26	70	M	28199/07	1	2	2	2	2	2	2	1	2	2	140/70	2	2	2	2	2	2	1+_	0.1

S.no	Age	Sex	Ip no	Oliguria	Fever	Dirrhoea	Vomiting	PE	Abd pain	Poisoning	Snake bite	DM	HTN	BP	Fever	Jaundice	PE	Facial puffiness	Breathlessness	Altered sensorium	urine alb	PCR
27	17	M	36681/07	1	1	2	1	2	2	2	2	2	2	120/80	1	2	2	1	1	1	1+	
28	60	M	22791/07	1	2	1	1	1	2	2	2	2	2	150/80	2	2	1	2	2	2	4+	
29	75	M	30690/07	1	1	2	1	2	2	2	2	1	1	180/110	1	1	1	1	1	1	5+	
30	33	F	31528/07	1	2	1	1	2	2	2	2	2	1	150/90	2	2	1	1	2	2	2+	
31	20	M	22771/07	1	2	1	1	2	2	2	2	2	2	110/80	2	2	2	2	2	2	3+	
32	55	F	25787/07	1	1	2	2	1	1	2	2	2	2	140/100	1	2	1	1	1	2	5+	2.2
33	34	M	21268/07	1	2	1	1	2	2	2	2	2	2	130/100	2	2	2	2	2	2	3+	
34	46	M	30326/07	1	1	2	1	2	2	2	2	2	2	50/?	1	2	2	1	1	1	2+	1.4
35	65	F	39001/07	2	2	1	2	1	2	2	2	2	1	160/90	2	2	2	1	1	2	3+	
36	55	F	30938/07	2	1	2	2	2	2	2	2	1	1	160/100	1	2	1	1	1	1	2+	
37	35	F	35781/07	1	2	1	1	1	2	2	2	2	2	140/90	2	2	1	1	2	2	1+	
38	26	M	28167/07	1	1	2	2	2	2	2	2	2	2	110/80	1	2	1	1	2	2	3+	
39	40	F	34970/07	1	1	2	2	1	1	2	2	2	2	120/80	1	2	1	1	2	2	1+	0.6
40	46	M	24725/07	1	2	2	2	2	1	1	2	2	2	150/70	2	2	1	2	2	2	3+	0.3
41	25	F	39735/07	1	2	1	1	1	2	2	2	2	2	120/80	2	2	2	1	2	2	3+	
42	66	M	35776/07	1	2	1	1	2	1	2	2	2	1	150/90	2	2	1	1	2	2	2+	
43	40	F	32789/07	1	2	1	1	1	1	2	2	2	2	110/90	2	2	1	1	2	2	2+	
44	20	M	24584/07	1	2	2	2	1	2	2	2	2	2	140/80	2	2	1	1	1	2	2+	
45	21	M	25523/07	2	2	1	1	2	1	2	2	2	2	120/100	2	2	2	1	2	2	1+	
46	23	F	30616/07	1	1	2	1	2	2	2	2	2	2	130/80	1	2	2	2	2	2	2+	
47	19	M	22801/07	1	2	1	2	2	2	2	2	2	2	160/80	2	2	2	2	2	2	2+	
48	85	F	11108/07	1	1	2	2	2	2	2	2	1	2	60/?	1	2	1	1	1	1	3+	
49	23	M	29813/07	1	2	2	1	2	1	1	2	2	2	150/70	2	2	2	2	2	2	5+	0.7
50	60	M	39753/07	1	2	1	1	2	1	2	2	2	2	140/90	2	2	2	2	2	2	1+	

S.no	Age	Sex	Ip no	Oliguria	Fever	Dirrhoea	Vomiting	PE	Abd pain	Poisoning	Snake bite	DM	HTN	BP	Fever	Jaundice	PE	Facial puffiness	Breathlessness	Altered sensoriu	urine alb	PCR
51	17	F	35509/07	1	2	1	1	2	1	2	2	2	2	100/60	1	2	2	2	2	2	2+	
52	30	M	39644/07	1	1	2	2	2	1	2	2	1	2	150/80	1	2	2	1	2	2	1+	
53	41	F	36918/07	1	2	1	1	2	1	2	2	2	2	140/100	2	2	2	2	2	2	4+	
54	17	F	34080/07	1	1	2	1	1	1	2	2	2	1	180/100	1	2	2	2	2	2	4	
55	76	F	39185/07	1	2	1	2	2	2	2	2	2	2	140/80	2	2	2	1	2	2	1+	
56	24	M	39741/07	2	2	2	1	2	1	1	2	2	2	130/80	2	2	2	2	2	2	3+	
57	60	M	39692/07	1	1	2	1	2	2	2	2	1	2	140/100	1	2	1	1	1	1	2+	
58	46	M	2865/08	1	1	1	1	2	2	2	2	2	2	100/70	1	2	2	2	2	2	1+	
59	51	F	25571/08	1	2	1	1	2	1	2	2	2	2	120/70	2	2	1	1	2	2	3+	
60	43	M	36585/07	1	1	2	1	2	2	2	2	2	2	150/80	1	1	2	1	2	2	1+	
61	45	M	2926/08	1	2	1	1	2	2	2	2	2	2	140/70	2	2	2	1	2	2	4+	
62	50	M	2932/08	1	1	2	1	2	2	2	2	2	2	136/70	1	2	1	1	2	2	3+	
63	48	M	43125/07	1	1	2	1	2	1	2	2	2	2	140/100	1	1	1	1	2	2	3+	
64	13	M	3819/08	2	1	1	1	2	2	2	2	2	2	130/100	2	2	1	1	2	2	4+	
65	54	M	1781/08	1	2	1	2	1	2	2	2	2	2	150/90	2	2	2	2	2	2	2+	
66	39	F	8070/08	1	1	2	2	2	1	1	1	2	2	170/100	1	2	1	1	1	2	4+	2.2
67	45	M	6306/08	1	2	1	1	2	1	2	2	2	2	130/70	2	2	2	1	2	2	1+	
68	46	F	4676/08	1	1	2	2	2	2	2	1	1	2	160/80	1	2	2	2	1	1	2+	
69	58	M	9772/08	1	2	1	1	2	1	2	2	2	2	110/80	1	2	1	2	2	2	2+	
70	75	M	11547/08	1	1	2	1	2	2	2	2	2	2	110/60	1	1	1	2	2	2	3+	0.5
71	35	M	9775/08	1	2	1	1	1	1	2	2	2	2	140/100	2	2	1	1	2	2	2+	
72	26	M	5509/08	1	1	2	1	2	2	2	2	2	2	120/60	2	1	2	1	1	1	2+	
73	48	M	11390/08	1	1	1	1	1	1	2	2	2	2	140/70	2	2	1	1	2	1	1+	
74	30	M	9776/08	1	1	1	1	2	2	2	2	2	2	100/70	1	1	2	1	2	2	3+	
75	15	F	12660/08	1	2	1	1	2	1	2	2	2	2	110/80	2	2	2	1	2	2	3+	

S.no	Age	Sex	Ip no	Oliguria	Fever	Dirrhoea	Vomiting	PE	Abd pain	Poisoning	Snake bite	DM	HTN	BP	Fever	Jaundice	PE	Facial puffiness	Breathlessness	Altered sensoriu	urine alb	PCR
76	30	F	5474/08	1	1	2	2	2	2	2	2	2	2	150/110	1	2	2	2	2	2	1+	
77	20	F	12283/08	1	2	1	2	2	2	2	2	2	2	140/70	2	2	2	2	2	2	2+	
78	36	M	10189/08	1	2	1	2	2	2	2	2	2	2	160/90	2	2	2	2	2	2	2+	
79	55	M	7922/08	1	1	2	1	1	1	2	2	1	2	160/110	2	2	2	2	2	2	4+	1.5
80	55	F	20410/08	1	2	1	2	2	1	2	2	2	2	100/60	2	2	2	2	2	2	2+	
81	55	M	22117/08	1	1	2	1	2	1	2	2	1	2	150/80	1	1	1	1	2	2	4+	
82	48	F	24139/08	2	2	2	2	2	2	2	2	2	2	150/80	2	2	2	2	2	2	2+	
83	76	M	38243/08	1	1	1	1	1	1	2	2	1	2	160/90	1	2	1	1	1	1	3+	
84	75	F	34497/08	1	2	1	1	1	1	2	2	1	1	160/110	2	2	1	1	2	2	1+	
85	28	M	34653/08	2	2	2	2	2	2	2	2	2	1	160/100	2	2	2	2	2	2	nil	0.3
86	30	M	35401/08	1	2	2	1	2	1	1	2	1	2	130/70	2	2	2	2	2	2	3+	0.6
87	60	F	37440/08	1	2	1	1	2	2	2	2	2	2	140/80	1	2	1	2	2	2	2+	
88	13	F	37776/08	1	1	2	1	1	2	2	2	2	2	160/100	1	2	1	1	2	2	5+	2.6
89	65	M	45376/08	1	2	1	2	2	1	2	2	2	2	120/80	2	2	2	2	2	2	2+	
90	50	M	39900/08	1	1	1	1	2	1	2	2	2	2	130/100	1	1	1	2	2	2	3+	0.7
91	55	F	20395/08	1	1	1	1	2	1	2	2	2	2	140/90	1	2	1	2	2	2	1+	
92	22	F	41828/08	1	2	2	2	1	1	2	2	2	2	130/80	1	2	2	2	2	2	4+	
93	26	F	70102/08	1	1	2	1	1	2	2	2	2	2	110/70	1	2	1	1	2	2	5+	2
94	50	M	69250/08	1	1	2	1	2	1	2	2	2	2	130/100	1	2	2	2	2	2	1+	
95	70	M	39084/08	1	2	1	2	2	1	2	2	2	2	160/100	2	2	2	2	2	2	2+	
96	50	F	66653/08	1	1	1	2	1	1	2	2	1	2	140/90	1	2	1	1	1	1	3+	
97	64	M	39878/08	1	2	1	2	2	2	2	2	2	2	140/70	2	2	2	2	2	2	3+	
98	52	F	19449/08	2	2	2	2	2	2	2	1	2	2	130/70	2	2	2	2	2	2	2+	
99	30	F	41706/08	1	2	2	1	2	1	1	2	2	2	150/80	1	2	2	2	2	2	1+	
100	22	F	42426/08	1	1	2	1	2	1	2	2	2	2	160/90	2	2	1	1	2	2	3+	3.1

FENa	HB	S.no	TC	Platelets	Mp	MSAT	widal	urea	creatinine			Na+	K+	OTHERS			type of AKI	RIFLE
									first	max	final							
	9.8	1	4500	2.6	1	1	2	56	1.8	1.8	1	134	4.5				1	R
	10.8	2	6400	1.2	2	2	2	78	1.6	2	0.8	123	3.5	stool-e.coli+			1	R
	10	3	6700	3.4	2	2	2	108	1.5	1.8	1.1	145	3.9				1	R
1.3	5.9	4	11000	1.2	2	2	2	112	3.2	9.8	6.4	129	3	ABG- met.acidosis			2	F
0.9	8.6	5	8900	2.2	2	2	2	76	2.2	2.8	0.6	156	4.9	ABG- met.acidosis			2	I
	10.8	6	7000	2.6	2	2	2	88	1.2	2.8	1	145	3.6				1	I
	9.2	7	4800	2	1	2	2	70	1.8	1.8	1	138	4.2				1	R
	12	8	7300	3.1	2	2	2	67	2.5	3	1.4	134	2.8	STOOL-EH+			1	I
	9.7	9	9600	2.8	1	2	2	68	1.5	1.8	0.9	135	3.7				1	R
0.6	9	10	5600	1.8	2	1	2	98	2	2	1	138	3.8	USG-N			1	R
	8.8	11	3200	1.6	2	2	2	67	1.1	5.6	9.2	156	2.2	ABG-met.acidosis	urine c&s- E.coli+		2	F
	10	12	7300	0.9	2	2	2	52	2.3	2.8	1.2	146	5.6				2	I
	10.8	13	6000	1.2	2	2	2	102	1.8	2.1	1.3	134	3.4	stool-e.coli+			1	I
1.5	7.9	14	13000	0.5	2	2	2	45	0.8	4.5	1.4	148	4.2	CXR-rt lower lobe pneumonia		USG-N	2	F
0.4	11.9	15	5100	2.3	2	2	2	96	3.2	4	1.1	138	5.2				1	F
	6.7	16	4100	3.6	2	2	2	67	2.8	3.4	3.4	110	6.8				2	I
	12.3	17	4800	3.6	2	2	2	92	6.7	8.9	6.8	138	6.7				2	F
	10.2	18	8700	4.1	2	2	2	83	1.5	1.8	1	135	4.4	stool- e.coli+			1	R
	9.4	19	5100	2.3	2	1	1	120	2.3	2.8	1.1	156	4	USG-N			1	I
	8.6	20	7300	2	2	2	2	82	6.2	7.8	1.6	130	5.6	USG-BPH			3	F
	10	21	9000	1.3	2	2	2	96	3	5.6	1.2	148	4.8	USG-N			1	F
	13	22	6900	3.2	2	2	2	56	1.1	2	1	130	3				1	R
1.5	9.9	23	14500	0.95	2	2	2	81	1	4.7	4.4	136	4.3	USG-N			2	F
	11.6	24	7700	1.8	2	2	2	94	1.6	1.6	0.9	135	4.5				1	R
	12	25	6500	3.2	2	1	2	62	2.2	2.8	1.1	134	3.8				1	I
1	10	26	9500	0.8	2	2	2	78	0.8	1.6	0.9	140	5	CT-20 MIN			2	R

FENa	HB	S.no	TC	Platelets	Mp	MSAT	widal	urea	creatinine			Na+	K+	OTHERS			type of AKI	RIFLE
	9.2	27	5000	3.4	1	2	2	60	4.2	8.3	1.4	142	4.7	ABG- resp.alkalosis			2	F
1.2	10.4	28	10100	2.2	2	2	2	80	5.2	8.3	1.1	152	2.5	stool- giardia+		USG-N	2	I
2	8.6	29	4200	1.4	2	2	2	58	3.2	9.2	9.2	156	5.3	USG-N			2	F
	9.6	30	5800	1.8	2	2	2	108	1	1.6	0.8	143	4.8				1	R
	11.8	31	4600	2.2	2	2	2	114	6.2	8.6	1.3	148	3.4	stool occult bld+			2	F
	8.8	32	9300	1	2	2	2	54	2.4	2.8	1.3	128	3.4	USG-N			2	I
	10	33	4000	3.8	2	2	2	82	1.6	1.6	0.6	138	5.7				1	R
1.4	9.6	34	9600	1.2	1	2	2	67	4.5	7.8	5.6	140	4.5				2	F
	9.8	35	4800	3.2	2	2	2	98	1.5	1.8	1	138	5.6				1	R
	12.5	36	12100	4	2	2	2	68	1	6.6	6	146	3.7	BLD C&S- staph+	ABG-met.acidosis		2	F
	10	37	5800	3.5	2	2	2	94	1	2.5	1	142	3.5				1	I
	12	38	8300	2.8	1	1	2	84	1.2	1.7	0.6	127	4.1				1	R
0.8	9	39	4300	2.3	2	2	1	98	3.4	4.6	1.1	132	4.3	USG-N			1	F
1	11.2	40	5000	1.9	2	2	2	45	1.8	2.3	1	138	2.8	USG-N			2	I
0.2	8.9	41	7200	1.5	2	2	2	98	3.4	4.5	1.2	126	3.8				1	R
	11.9	42	5900	3	2	2	2	86	1.4	1.6	0.7	150	3.4	STOOL- e.coli+			1	R
	9.8	43	9100	2.7	2	2	2	56	1.8	1.8	1.1	132	4.9				1	R
1.4	10	44	4300	1	2	2	2	48	2.3	3.4	1.2	134	4.5	CT-15 MIN			2	F
	11	45	7300	2	2	2	2	97	1.2	1.8	0.8	143	3.8				1	R
	9	46	6800	1.7	1	2	1	56	1.8	1.8	0.7	134	3.5				1	R
	10.3	47	7900	3.2	2	2	2	50	1.4	1.6	1	142	5.4				1	I
	9	48	9900	1	2	2	2	78	0.7	7.8	8	123	6	BLD C&S- e.coli+			2	F
1	12.7	49	4500	2.3	2	2	2	67	3.2	8.9	1.2	150	3.8				2	F
	9.2	50	5900	2.3	2	2	2	50	0.7	1.4	0.7	138	4.1	stool-enterobius ver			1	R

FENa	HB	S.no	TC	Platelets	Mp	MSAT	widal	urea	creatinine			Na+	K+	OTHERS			type of AKI	RIFLE
	12.8	51	8700	3.7	2	2	2	68	1.5	1.5	0.8	144	5.6				1	R
	10.3	52	10200	2.6	1	1	2	89	1.4	2	0.6	148	3.4	USG-N			1	R
	11	53	6000	1.3	2	2	2	80	5	8.2	1.2	152	6.7				1	F
	8.4	54	3700	0.9	2	1	2	100	1	1.9	1	138	4.3	USG-N			1	R
	12.4	55	6300	1.8	2	2	2	98	1.8	1.8	1.1	134	4.7	stool-e.coli+			1	R
1.2	9.5	56	5200	2.8	2	2	2	68	1.4	2.2	0.6	140	3.4				1	I
	10.5	57	9200	4.2	2	2	2	134	0.9	8.2	4.2	130	4.2				1	F
	12	58	6700	3.8	2	2	2	96	1.8	1.8	0.7	128	4.8	stool-giardia+			1	R
	9.2	59	10000	2.6	2	2	2	73	1.4	1.9	0.8	146	5.2	stool-giardia+			1	R
	10	60	6400	1.1	1	2	2	93	2.8	2.8	1.3	152	3.4				1	I
	12.4	61	4500	2.7	2	2	2	106	1.4	1.7	1	138	5.8				1	R
	11.6	62	5600	3.6	2	2	1	58	1.5	1.8	1	127	4				1	R
0.2	7.2	63	9200	1.3	1	1	2	82	1.3	1.9	0.6	150	3.2				1	R
	11.8	64	5800	1.8	2	2	2	48	1.4	1.7	0.9	148	2.2				1	F
	10.5	65	3900	3.8	2	2	2	84	1.8	1.8	1.1	134	5	stool-EH+			1	R
1	10.8	66	9700	1.1	2	2	2	45	1.6	2.4	0.9	146	4.6				2	I
	11.7	67	8400	1.4	2	2	2	54	1.6	1.6	0.8	144	4.8				1	R
1.6	9.4	68	6000	0.95	2	2	2	1.8	1.8	5.6	1.2	136	4.5	USG-N			2	F
	13.6	69	4800	4.6	2	2	2	78	2.3	2.8	0.9	130	4	stool-e.coli+			1	I
0.6	11	70	11000	1.4	1	1	2	72	1.3	1.5	1	146	3.5				1	R
	10.8	71	6300	3.6	2	2	2	104	1.7	1.7	0.9	152	4.7				1	R
	9.2	72	4100	1	1	2	2	72	3.9	4.5	2.2	140	7.2				2	F
	10.2	73	5000	3.9	2	2	2	102	1.8	1.8	1	154	4.2	CSF-sugar-20 mg, p			1	R
	9.6	74	8200	3.1	1	1	2	86	1.5	1.9	0.9	137	5				2	R
	9	75	7600	2.3	2	2	2	84	2.1	2.5	0.8	142	3.2	STOOL-EH+			1	I

FENa	HB	S.no	TC	Platelets	Mp	MSAT	widal	urea	creatinine			Na+	K+	OTHERS			type of AKI	RIFLE
	8	76	5200	3.8	1	2	2	75	2	2.5	1.5	156	4.8				1	I
	9.8	77	4800	2	2	2	2	67	1.6	1.6	1	130	4.8				1	R
	11.2	78	6900	2	2	2	2	48	1.5	1.9	1	135	2.6				1	R
1.2	9.4	79	3100	1.3	2	2	2	72	0.8	6.7	3.4	148	4.5	urine c&s- e.coli+	ABG-met.acidosis		2	F
	10	80	5800	3.3	2	2	2	88	1.7	1.7	0.8	139	3.4				1	R
	11.6	81	6800	3.7	2	1	2	96	1.8	1.8	0.8	144	4.6	USG-N			1	R
1	10.4	82	5700	2	2	2	2	69	0.8	2.8	0.9	156	4.3	USG-N	RF+		2	I
1.5	10.2	83	13600	2.3	2	2	2	70	0.7	4.3	1	149	4.5	sputum c&s- klebse			2	F
	11.2	84	8900	2.5	2	2	2	98	1.5	1.5	0.6	130	5.3	USG-N			1	R
	10	85	8300	3.5	2	2	2	26	1	1.7	1	134	5.3	USG-N			1	R
0.7	10	86	7000	4.2	2	2	2	56	1.5	2.6	1.2	140	3.7	USG-N			1	I
	11	87	7800	3	2	2	2	110	1.6	1.8	0.7	128	3.1				1	R
1.3	9.2	88	9000	3.2	2	2	2	49	2	2.9	1.2	146	3.5	USG-N			2	I
	8.5	89	6200	2.2	2	2	2	90	1.4	1.8	0.8	155	4.8				1	R
1.1	9	90	9400	0.5	1	1	2	78	1.7	1.7	1	145	2.8				2	R
	7.8	91	3400	0.8	1	2	2	66	3	9.7	4.9	129	4.9	ABG-metabolic acid			1	F
	11	92	6700	3.1	2	2	2	107	1.6	2.3	1	127	3		stool- giardia+		1	I
	10.3	93	4500	2.1	2	2	2	68	2.5	2.9	1.3	142	5.6	USG-N			2	I
	8.4	94	8400	2	2	2	2	74	2.2	2.2	1.8	129	3.1	USG- b/l staghorn	calculi		3	I
	8.9	95	7800	1	2	2	2	100	1.6	1.6	0.8	149	4.2				1	R
	10	96	3800	1	2	2	2	64	4.5	7.4	7	125	2.9	BLD C&S- strepo. V			2	F
	10.6	97	7300	2	2	2	2	109	1.6	1.9	0.7	138	4.8	STOOL-EH+			1	R
	9.6	98	12000	0.8	2	2	2	71	3.4	5.4	2.3	142	6.4		CT- >15 min		2	F
1.2	11.4	99	6000	3.6	2	2	2	60	1.8	4.5	1	152	4.3	ABG-met. Acidosis			2	F
	11.4	100	6500	1	2	2	2	67	1.6	2.1	0.8	136	3.4				2	I

treatment
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treatment
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treatment
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S.no	complications		Diagnosis		expired(days()	
1			LEPTOSPIROSIS/MALARIA			
2			ADD			
3			ADD			
4	metebolic acidosis		UNKNOWN POISON		2	
5	metebolic acidosis		CUSO4 POISON			
6			ADD			
7			MALARIA			
8	pulmonary edema		ADD			
9			MALARIA			
10			LEPTOSPIROSIS			
11	metebolic acidosis		SEVERE SEPSIS		2	
12			SNAKE BITE			
13			ADD			
14			SEVERE SEPSIS			
15			ADD			
16	arrythmias		ADD			
17	arrythmias		ADD		2	
18			ADD			
19	pulmonary edema		LEPTOSPIROSIS/ENTERIC FEV			
20			BPH			
21			ADD			
22			UNKNOWN POISON			
23	hypotension		SEPSIS/ SHOCK		1	
24			ADD			
25	ALTERED MENTAL STATUS		LEPTOSPIROSIS			
26			SNAKE BITE			

S.no	complications		Diagnosis		expired(days()	
27			CEREBRAL MALARIA			
28	pulmonary edema		ADD			
29			SEPSIS/ MODS		4	
30			ADD			
31	GI HEMORRHAGE		ADD			
32			NEPHRITIC SYNDROME(PIGN)			
33			ADD			
34	hypotension		ALGID MALARIA		1	
35			ADD			
36	metebolic acidosis		SEPSIS/ MODS		3	
37			ADD			
38			LEPTOSPIROSIS/MALARIA			
39			ENTERIC FEVER			
40			CUSO4 POISON			
41			ADD			
42			ADD			
43			ADD			
44	ALTERED MENTAL STATUS		SNAKE BITE		2	
45			ADD			
46			MALARIA/ ENTERIC FEVER			
47			ADD			
48	hypotension	hyponatremi	SEPSIS/SHOCK		2	
49			PARACETAMOL POISONING			
50			ADD			

S.no	complications		Diagnosis		expired(days()	
51			ADD			
52			LEPTOSPIROSIS/MALARIA			
53	hyperkalemia		ADD			
54			LEPTOSPIROSIS			
55			ADD			
56			CUSO4 POISON			
57	pulmonary edema		SEVERE SEPSIS		2	
58			ADD			
59			ADD			
60			MALARIA			
61			ADD			
62			ENTERIC FEVER			
63			LEPTOSPIROSIS/MALARIA			
64	hypokalemia		ADD			
65			ADD			
66			NEPHRITIC SYNDROME			
67			ADD			
68			SNAKE BITE			
69			ADD			
70			LEPTOSPIROSIS/MALARIA			
71			ADD			
72	arrythmias		CEREBRAL MALARIA		2	
73			TB MENINGITIS			
74			MALARIA/ LEPTOSPIROSIS			
75			ADD			

S.no	complications		Diagnosis		expired(days()	
76			MALARIA			
77			ADD			
78			ADD			
79	metebolic acidosis		SEPSIS/MODS		4	
80			ADD			
81			LEPTOSPIROSIS			
82			DRUG INDUCED (NSAIDS)			
83	hiccups		SEVERE SEPSIS			
84			ADD			
85			DRUG INDUCED (ACEI)			
86	metebolic acidosis		DICHROMATE POISONING		2	
87			ADD			
88			NEPHRITIC SYNDROME(PIGN)			
89			ADD			
90			MALARIA/ LEPTOSPIROSIS			
91	metebolic acidosis		MALARIA			
92			ADD			
93			NEPHRITIC SYNDROME			
94			RENAL CALCULUS			
95			ADD			
96	hypotension		SEPSIS/ SHOCK			
97			ADD			
98	arrythmias	hyperkalemia	SNAKE BITE			
99	metebolic acidosis		DICHROMATE POISONING			
100			NEPHROTIC SYNDROME			

ABBREVIATIONS-MASTER CHART

s. no – serial number

Ip.no – In patient number

PCR- Protein creatnine ratio

FENa- Fractional excretion of sodium

MP- Smear for malarial parasite

MSAT- Macroscopic slide agglutination test

ABG- Arterial blood gas analysis

USG-N- Ultrasonogram of kidney was normal study

CT- Clotting time

C&S – Culture and sensitivity

CXR-Chest X-ray PA view

ADD- Acute diarrhoeal disease

CSF- Cerebrospinal fluid

EH- Entamoeba histolytica

Others- Other investigations

Oliguria- Hypertension

1- yes

2-no

Fever-Alterde sensorium

1- yes

2-no

MP, MSAT, WIDAL

1- Positive

2- Negative

Type of AKI

1- Prerenal

2- Renal

3-Postrenal

RIFLE

1- RISK

2-INJURY

3-FAILURE

Treatment

1-Conservative

2-Peritoneal dialysis

3-Hemodialysis